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Proceedings of conference of collaborators from Southern Agricultural Experiment Stations Agricultural research and consumer health safety, March 20 and 21, 1972

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RESEARCH DIVISION

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PREFACE

This Conference was sponsored jointly by the Southern Agricultural Experiment Stations and Southern Marketing and Nutrition Research Division of Agricultural Research Service, U.S. Department of Agriculture. Its purpose was to exchange information that directly benefits the Stations, the Divisions of the Agricultural Research Service, and others.

The theme for the 1972 meeting was "Agricultural Research and Consumer Health and Safety." There were four sessions — the first dealing with toxicants and their elimination; the second, with control of health problems; the third, with safety; and the fourth, with consumerism.

These proceedings report the statements presented by the various speakers during the conference.

C. H. FISHER, Director
Southern Marketing and Nutrition
Research Division

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Underscored numbers in parentheses refer to references at the end of each paper. References, figures, and tables are reproduced essentially as they were supplied by the author of each paper.

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PROCEEDINGS OF THE CONFERENCE OF COLLABORATORS FROM SOUTHERN AGRICULTURAL EXPERIMENT STATIONS
AGRICULTURAL RESEARCH AND CONSUMER HEALTH SAFETY
MARCH 20 AND 21, 1972

WELCOME

by
C.H. Fisher, Director
Southern Marketing and Nutrition Research Division
New Orleans, La.

On behalf of the Southern Marketing and Nutrition Research Division (SMNRD), I welcome you to this Collaborators' Conference on Agricultural Research and Consumer Health Safety.

The practice of holding annual Collaborators' Conferences started more than 30 years ago. We at SMNRD continue to be grateful for the privilege of sponsoring these annual conferences in cooperation with the Directors and other administrators of the Southern Agricultural Experiment Stations. We appreciate also the opportunity of working with State and other organizations to benefit agricultural research and those who produce, process, and consume agricultural products.

In recent years, these annual conferences have given increasing attention to major problems of concern to farmers and consumers alike. Last year's conference, for example, featured talks on "Agricultural Research and Pollution Abatement." That conference made it abundantly clear that we in agricultural research are doing great things to improve our environment. A principal purpose of the present conference is to review what agricultural research has done — and is doing — for consumer health and safety.

I wish to bring to your attention several changes and events that have occurred during the past year. We now have a more flexible patent and licensing policy. As before, nonexclusive licenses may be granted for the practice of our inventions. Under some conditions, exclusive licenses may be granted. For the last several years, about 50 patents per year have been granted to inventors of SMNRD.

Two laboratories formerly associated with SMNRD have been made a part of our sister Southeastern Marketing and Nutrition Research Division, which is headquartered at Athens, Ga. One is the Fruit and Vegetable Products Laboratory, Winter Haven, Fla., and the other is the Food Fermentation Laboratory, which works in cooperation with the North Carolina State University and Agricultural Experiment Station, Raleigh, N.C.

The Textiles and Clothing Laboratory, Knoxville, Tenn., has been made a part of SMNRD. We are fortunate because Mary E. Carter, a distinguished textile scientist, joined SMNRD as Chief of the Textiles and Clothing Laboratory. The Textiles and Clothing Laboratory is located on the campus of the University of Tennessee, and we are grateful for the privilege of working in cooperation with the University of Tennessee, particularly its Department of Home Economics.

During the past year, our research, conducted in cooperation with State, industrial, and other organizations, continued to be profitable. Some of our research findings have been adopted by other organizations and used for practical purposes, and some of our developments that were previously commercialized are being used more extensively.

Our scientists and engineers, working cooperatively with other organizations, have contributed importantly to 15 commercialized developments during the past year. A number of these are directly related to consumer health and safety.

The Liquid Cyclone Process pilot plant at Sundatta Oil Mill, Hubli, India (Dorr-Oliver, Inc.) is producing an edible cottonseed flour concentrate, and the capacity of the plant will be increased from 1 to 5 tons per day. A Liquid Cyclone Process plant for producing 25 tons of high quality protein flour from glanded cottonseed is under construction at Plains Cooperative Oil Mill, Lubbock, Tex. The plant is scheduled to begin production in 1972. The product, which contains about 70 percent nutritious protein, has the capability of improving human health.

The Millicolumn method for detecting and measuring toxic aflatoxins in cottonseed and peanuts in about 15 minutes is being used with both dollar and health benefits.

Although a number of flame-retardant formulations containing THPC are used commercially, two new products are now offered by American Cyanamid, their first entry into the THPC area. One of the products, TKC, is a 60-percent solution of THPC. The other product is TKP, an adduct of THP and urea. These products are used to protect consumers by imparting flame resistance to textile products.

Citrus World, Inc., Lake Wales, Fla. (a cooperative), is manufacturing orange juice solids or crystals. They are reported to be packing more than 555,000 15-1/2 oz. cans of crystals for the Department of Defense, with plans to begin packaging for the retail trade soon. The process is based on cooperative research by our sister Western Marketing and Nutrition Research Division, our former Fruit and Vegetable Products Laboratory, Winter Haven, Fla., and the Florida State Department of Citrus. Although you did not learn this from TV programs, orange juice crystals were aboard our Apollo flights 13, 14, and 15.

Progress in our research in Calendar Year 1971 was reported in 298 technical papers, 175 presentations to scientific and industrial groups, and 63 patent applications. Eight patent licenses were granted. Four honor awards were received by our scientists.

For any successes we have had we are indebted in part to our friends in State, industrial, and other organizations. A considerable part of our cooperative research on cottonseed and linters is supported by funds from the National Cottonseed Products Association, National Cotton Batting Institute, and Cotton Incorporated. We are grateful for this encouragement and support.

I wish to thank everyone contributing to the success of this conference. In particular, I wish to thank Drs. Doyle Chambers and H. Rouse Caffey of the Louisiana State Stations, the Program Committee, our General Chairman, Session Chairmen, the speakers, our visitors, and the staff of the Southern Marketing and Nutrition Research Division for the arrangements that have made this conference possible.

I hope that your stay in New Orleans will be enjoyable and rewarding. Please visit our Laboratory and talk with our scientists and engineers. We shall be glad to have suggestions that will help us make our program more valuable to agriculture and all consumers.

FEDERAL RESEARCH IN AGRICULTURE IMPROVES HUMAN HEALTH AND SAFETY

by
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Agricultural Research Service
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Washington, D.C.

According to present plans, a new satellite will be launched into orbit in June. It is the unmanned Earth Resources Technology Satellite — ERTS — and with its launching, agricultural science will be projected into a new orbit. For a year, this experimental satellite will circle the earth, testing its multisensing instruments to see if, from 500 nautical miles in space, they can transmit to us a running account of the state of many of the world's resources.

In the U.S. Department of Agriculture, we want to see if we can monitor soil and water conditions throughout the growing season . . . identify crops and their state of health . . . distinguish between good and poor cropland . . . and determine the state of watersheds and of forests — all on an international basis. Think what such remote sensing — if successful — would mean toward growing better and more nutritious crops and livestock for a hungry world!

The implications for bettering the health and safety of future generations are incalculable. And so are those of another bit of recent agricultural research — not in outer space, but in the depths of inner space.

Last August USDA's Agricultural Research Service announced that a new kind of disease-producing particle, smaller than a virus, had been isolated and identified by one of our pathologists, Dr. Theodor O. Diener. He calls the new pathogen, which causes the spindle tuber disease in potatoes, a "viroid." He is convinced that many plants, animals, and human diseases whose causes have eluded the probing of science are probably caused by viroids — such diseases of the central nervous system as scrapie of sheep, kuru — a fatal degenerative disease of natives of the New Guinea highlands, and even multiple sclerosis, infectious hepatitis, and possibly some types of cancers.

These are examples of the widely varied research in agriculture that holds potential for human health and safety.

Such research has, of course, provided us with better ways to produce and deliver wholesome, abundant food to consumers at relatively low cost. Production research enables farmers to produce more for less . . . through the development of high-yielding crop varieties and livestock breeds . . . and through mechanization.

Marketing research has speeded nutritious food to consumers through improved handling methods, better marketing facilities, packaging, transport, and storage. Marketing specialists have designed wholesale food markets for most major cities in the United States — and even for London and Paris — that help move food fresher, cleaner, and faster from farms to dinner tables — and at lowered handling costs. Research has also provided standards and testing equipment to improve methods for determining quality and detecting impurities during food grading and inspection.

New food products and processes developed and improved through research have given grocery shoppers wider choices for supplying needed nutrients to their families in more varied and more delicious forms.

Nutrition research has been applied to identify and help preserve in our food supply the vitamins, proteins, and minerals that are essential to our health and well-being. ARS research has also given us new concepts of human nutritional needs and of adequate diets for all age groups.

But our research has done much more than identify and provide protective foods.

In combating the pathogens and hazards that beset plants and animals, USDA scientists over the years have devised means to prevent, diagnose, and treat many human ills.

THE PAST IS PROLOGUE.

Early in the 1890's, for example, Theobald Smith and F.L. Kilborne solved the mystery of Texas cattle fever with the discovery that a tick could transmit disease. From this basic discovery stemmed the control of yellow fever and many other pest-borne human diseases.

Later, during World War II, scientists forged efficient weapons against the mosquitoes, lice, ticks, and fleas that carry diseases such as malaria, typhus, encephalitis, and bubonic plague. This research has saved millions of lives and prevented hundreds of millions of illnesses around the world.

Other USDA scientists discovered the New World hookworm, and evolved methods of preventing spread of trichinosis and beef tapeworms to humans. In 1969, ARS research verified a method of detecting trichinosis. It is conceivable that this method could lead to the eventual eradication of trichinosis in the Nation's swine.

ARS scientists also developed a vaccine for brucellosis that made possible the ARS-State campaign to eradicate brucellosis from cattle and hogs. This had dramatically reduced cases of undulant fever, the form the disease takes in humans.

A nationwide, cooperative attack on tuberculosis in dairy cattle was made possible when scientists developed methods of testing cattle for the disease. Before this campaign, hundreds of children died annually of tuberculous meningitis and miliary tuberculosis contracted from cow's milk or other contacts with infected cattle; other children who survived the diseases lived out their lives as hunchbacks.

More recently, the discovery that one form of poultry cancer can be transmitted through bird-to-bird contact gives added support to scientists who believe that viruses may cause some kinds of cancer. In another project, scientists found a new source of an enzyme that inhibits leukemia. Our plant explorers, meanwhile, have been combing the world for plants containing substances with a potential for treating cancer. From their promising search, which the National Cancer Institute supports, may come new weapons against the disease and new crops for American agriculture.

The discovery of antibiotic-producing molds changed medical practices, giving doctors a whole new armory of weapons against infections. ARS scientists discovered high-yielding organisms and developed fermentation processes for the mass production of penicillin that greatly reduced its cost and marked the beginning of the antibiotics industry.

Other contributions of agricultural scientists to human medicine include: Processes for producing dextran, a vital blood extender for victims of shock. The identification of many plant sources for cortisone, which has a dramatic remedial effect on rheumatoid arthritis. A risk-free and highly specific test for allergy that has implications for future medical practice and research. A simple and sensitive test for diagnosing galactosemia, a rare but severe metabolic disease of infants. Still under development is a high-calorie cottonseed oil emulsion for patients who require extended intravenous feeding.

A new process for tanning sheepskins makes them ideal padding for preventing and curing bedsores in bedridden patients. Cotton stretch bandages developed by research contribute to a patient's comfort.

ARS home economists and microbiologists have shown consumers how to make sure that disease-causing bacteria are destroyed in automatic washing machines and dishwashers. Housing specialists have designed energy-saving kitchens to conserve the health of handicapped, ill, or elderly women . . . fallout shelters to protect the lives and health of families in case of danger from atomic radiation . . . and means of handling soil and removing radiostrontium from milk in the event of a nuclear emergency.

Flame-retardant paints, cotton, and cotton fabrics developed here in New Orleans hold the potential for saving countless lives and preventing painful injuries, as well as for reducing many economic losses from fire. The chief of the Washington, D.C., fire department once said that when his firemen started wearing fire-retardant cotton clothing, they could get in much closer to the sources of fires and thus put them out much more quickly.

In 1964, the Surgeon General of the Public Health Service published his report on smoking and health. Since 1965, more than 60 percent of ARS research on tobacco, an important agricultural crop, has been health-related. ARS considers the search for a safe cigarette to be its responsibility. The scientists seek, first, to pinpoint substances in tobacco or tobacco smoke that are undesirable from the standpoint of human health and, second, to find ways to prevent the formation of such substances or to remove them.

Work is going on on other nonfood crops that affect human health — narcotic-producing plants. In cooperation with research institutions in foreign countries, our scientists are seeking to develop programs of research that would, first, provide suitable alternate crops and farming systems to substitute for areas taken out of narcotics production, and, second, provide methods for detecting illicit growth of narcotic-producing plants and destroying them without adverse ecological effects.

These ARS contributions will be augmented as scientists continue to close gaps in knowledge and raise the level of our well-being.

I certainly don't want to imply that ARS contributions to human health and safety outweigh those of agricultural research in the State experiment stations, in industry, and elsewhere. Credit goes to many, many scientists, organizations, and institutions for such accomplishments. Today, however, I think I'd better stick mostly to the USDA accomplishments with which I am familiar. Since much of our research is cooperative with a lot of other people, please consider that I am including them when I talk about ARS or USDA research.

Let's consider nutrition research first.

NUTRITION.

When it comes to diet, we have learned a great deal about how food affects the human body and why.

Forty years ago, hundreds were dying each year in the United States from pellagra and scurvy, both vitamin-deficiency diseases. The enrichment of white flour began during World War II. By the late 1950's, the major nutritional deficiency diseases were almost unknown in the United States and Canada. For awhile, we became so complacent about our nutritional knowledge and use of food technology and distribution that support for nutrition teaching and research was hard to come by.

Fortunately, we have realized that we are not so well off nutritionally. Anemia, and enlarged thyroids — the latter suggestion of iodine deficiency — are still with us, and marginal deficiencies are widespread. Research suggests that nutrition is involved in major problems such as some types of diabetes, arterial and heart diseases, and weight control. Too, we really don't know nearly enough about nutritional needs or how to deliver nutrition to those in need.

Nutrition can influence the development and degree of heart and vasculatory disease, which contributes to more than half the deaths in the United States. Specialists estimate that improved nutrition could reduce by 25 percent the number of people with heart disease under age 65, but new knowledge is needed to achieve this potential. Our nutritionists are contributing much to the vast amount of research needed before we know just what we should eat to favorably affect blood cholesterol levels. For example, some of our studies with experimental animals suggest that diets containing large amounts of simple sugars may have effects on blood lipids similar to those of fats.

Our animal scientists have developed leaner strains of swine and are working to modify or reduce the fats in beef, milk, and broilers. They expect to be able to reduce egg cholesterol by as much as 50 percent.

Nutritionists are increasingly aware that a number of new nutrients may prove essential to man.

Our new Human Nutrition Laboratory at Grand Forks, N. Dak., will be devoted mainly to research on trace minerals and to studies with human subjects. Scientists at Grand Forks and Beltsville, Md., hope to arrive at recommendations for diets that will assure people of all ages satisfactory and safe levels of available minerals.

Of the approximately 100 known elements, 20 have been proved essential to man. Among these are chromium — the importance of which was proved by one of our own scientists — and copper, iodine, iron, and zinc. There are indications that several more will be found indispensable to the human body. Our scientists recently demonstrated that nickel and vanadium are essential to the growth of chicks, and may prove equally essential to man. So may tin.

Our nutritionists and food economists provide the backup information to make the commodity distribution programs and the school lunch, breakfast, institutional, and head-start food programs as acceptable and nutritionally satisfying as possible.

ARS scientists develop high-protein child food supplements from soybeans and cereals and breed high-protein wheats for use in developing countries where most of the proteins must come from plants. Such foods can help to prevent deficiency diseases that can physically or mentally cripple hungry children.

Research in ARS has shown that fluid milk can be fortified with iron, and processing procedures to retard off-flavor development have been worked out. We believe such fortification is practicable on a large scale, and research on this is in the demonstration stage. ARS scientists have developed commercial methods for producing vitamins B_{12}, beta-carotene (pro-vitamin A), and riboflavin which make diet supplementation more practical.

The fortification of several cereal products with higher levels of iron, thiamin, riboflavin, and niacin is being considered. We need to give more consideration to the restoration and fortification of processed foods with vitamins and trace minerals. Restoration of nutrients, such as vitamin C in dehydrated potatoes, should be encouraged. We also need to monitor the development of new foods to insure that they are at least as valuable nutritionally as the foods they displace — and preferably better. We probably need to fortify snack foods, total packaged meals or main dishes, pastas, and other cereal foods.

Not everyone can drink milk, and we want to know why. Research has shown that some people, especially among adult blacks, Asians, and Indonesians, have trouble digesting lactose, the sugar in milk. We need to learn how to improve milk tolerance, especially in some children. Research is underway at our Eastern Laboratory on enzymic processes to split lactose in milk to galactose and glucose which are assimilable.

Other people, particularly infants placed on a diet of cow's milk at too early an age, sometimes become allergic to milk. One of our scientists has found that when a person drinks milk, new antigens are generated on digestion, all of them potential allergens. With this new knowledge, we may be able to devise methods of neutralizing the effect of these allergens in milk.

OVERT HAZARDS THAT THREATEN HUMAN HEALTH AND SAFETY.

Let's look now at some of the overt hazards that threaten human health and safety, and see what agricultural research is doing about them.

Dr. Virgil O. Wodicka, Director of the Bureau of Foods of the Food and Drug Administration, U.S. Department of Health, Education, and Welfare, has been quoted as listing the threats to our food supply in this order of importance: (1) microbial contamination; (2) nutritional imbalance, which we have already discussed; (3) environmental threats, such as traces of heavy metals in fish; (4) natural toxicants; (5) pesticide residues; and (6) food additives.

Microbial Contamination

According to a new book by Sally Smith Booth, Hung, Strung, and Potted — a history of eating habits in Colonial America — the "good old days" were really not all that good. Recipes describe how "to cure tainted fish" and "to recover venison when it stinks." It's no wonder that stomach disorders were common.

The most dangerous threat of all to our food supply is microbial contamination. Here, of course, is an area where agricultural scientists have been working for many years.

It is hard to realize that, early in this century, many people regarded pasteurized milk as unsafe for babies. It took years of painstaking work for a USDA bacteriologist to prove that pasteurized milk was safe even for babies and invalids to drink.

Since then, utilization scientists have developed a process for pasteurizing liquid egg white and developed technology that permits the use of pasteurized milk in cheese-making.

Through the years, ARS scientists have helped control Salmonella by setting sanitation guidelines for the processing of poultry, fish, and animal products. ARS has also helped develop test procedures for the isolation of Salmonella organisms from animal feeds and feed ingredients, poultry, and eggs, and other commodities. Scientists are now seeking to improve techniques for identifying salmonellae by using analytical rather than biochemical methods, and to find better ways of detecting trichinae in hog carcasses.

Our research has developed other analytical methods for detecting pathogenic microorganisms in food products, and practical methods of improving sanitation in fruit and vegetable processing plants. It has identified points of microbial contamination in red meat slaughterhouses and developed steam scalding of poultry to eliminate cross-contamination in scalding tanks.

Molds that Attack Food and Feedstuffs

Our scientists are also looking for new ways to control toxins produced by molds that attack maturing crops and food and feedstuffs during processing and storage.

Over the centuries, the ubiquitous molds have been responsible for massive outbreaks of mycotoxicosis in human beings. But only in the past decade did the acute toxicity and carcinogenic properties of aflatoxin become known. Then we realized that relatively low levels of mycotoxin contamination in foods could present serious health hazards. The capacity of the molds to produce toxins and carcinogens adds a new dimension and a new urgency to research.

Research has produced a wealth of information about aflatoxin in the last 10 years. Sterigmatocystin, a toxic metabolite of Aspergillus versicolor, has also been isolated in fodder and peanuts, and should receive more attention as a public health hazard. Many other toxic mold metabolites have been isolated and identified. The ochratoxins are new fungal metabolites isolated from corn cultures of Aspergillus species. Isolation of ochratoxin and another toxin, citrinin, from Penicillum viridicatum has also been reported recently.

The list of fungal toxins goes on and on. Methods for detecting and estimating fungal toxins are essential.

Such methodology has been developed and employed for the control program instituted by the Department in cooperation with FDA and the peanut industry, to keep peanuts contaminated with aflatoxins out of the food marketing system. The control program works well to provide an adequate supply of wholesome peanuts for consumers, but the problem of control at the source point of infection has not been solved.

Aflatoxin contamination has been found in cottonseed, corn, and tree nuts. ARS scientists have recently developed a rapid "millicolumn" analytical method for detecting aflatoxin that has been adopted by the cottonseed industry. Our scientists have applied this method to corn. Other scientists have developed the use of ultraviolet radiation for detecting aflatoxin in corn, and processing methods to inactivate aflatoxin or remove it from oilseed meals.

The recent epidemic of corn blight highlights another important problem that must increasingly receive attention — secondary mold infection.

We are concerned lest some secondary invaders that come along with or follow blight may be toxic to animals and man. When dealing with toxins active in the parts-per-billion range, we must look to the improvement of our present agricultural practices, both on and off the farm.

Environmental Contamination

We are more concerned about the cumulative effect in our bodies of many substances that enter our foods than about any immediate dangers.

Industrial chemicals like PCB's — polychlorinated biphenyls — have been in the news a lot lately. They have gotten into poultry and animal feeds accidentally, but they are also present in low background levels almost everywhere in the food supply. Our scientists have determined how PCB in paint used on inside silo walls gets into feed and thus into cows and their milk, and are working out practical ways of cleaning up the problem.

Some heavy metals also get into our food; for example, mercury contamination of fish is of widespread concern. Mercury can be recovered from contaminated water by wool, and ARS utilization scientists are conducting research to determine whether this use of wool has practical value.

Another scientist has learned how to determine the concentration of cadmium and lead in dairy products with extreme accuracy. He is starting research to discover what correlation, if any, exists between the cadmium and lead content of cows' diets and that of the milk they produce.

Natural Toxicants

During the centuries of man's existence, he has learned to reject food of poisonous plant species and has selected other, more innocuous species to develop and domesticate. Yet even these foods are not necessarily perfect for our needs, and frequently contain harmful or potentially harmful substances.

Scientists have succeeded in breeding out of cotton the glands that contain gossypol, a naturally occurring toxin that has limited the use of cottonseed meal as feed for nonruminant animals or in human food. More immediately useful is the liquid cyclone process, developed at our laboratory here, for preparing cottonseed flour fractions high in protein and very low in gossypol.

Vegetables and condiments of the plant family that includes cabbage, turnips, rutabaga, and mustard contain substantial quantities of compounds called thioglucosides, or glucosinolates, some of which are strongly goitrogenic.

The thioglucoside content varies among the leafy and root vegetables that people eat, but varieties have not been specifically developed that minimize quantities of goitrogens. Seed meals from cabbage family plants used as animal feed require special processing to reduce the goitrogenic and other effects of the naturally occurring thioglucosides present.

Other types of naturally occurring goitrogens have been reported in soybeans and several other common vegetables.

It has long been known that the white potato could contain toxic amounts of solanine, a glycoalkaloid. Since breeders often look to other plant species for sources of resistance to disease or insects, surveys are being made of the solaine content of both older potato varieties and breeding materials.

Where resistance to insects or diseases has a chemical or biochemical basis rather than a physical basis, breeders of all food crops should be aware of the possibility of tailoring new varieties to be pest resistant and inadvertently making them, at the same time, toxic to animals or humans.

The Food and Drug Administration now requires appraisal of the safety of newly developed varieties of food plants where breeding or selection may reasonably be expected to alter significantly their nutritive value or the concentration of toxic constituents in them.

Undoubtedly, the biological activity of a host of other chemical compounds that plants contain in small concentrations will receive research attention in coming years.

Pesticide Residues

Our entomologists in ARS have for years been engaged in a massive research effort to reduce use of persistent pesticides and to find alternate pest control methods, so as to keep harmful chemicals out of the food chain for fish, birds, and animals as well as humans. Pest-resistant crops are being developed, and selective natural parasites, predators, and pathogens are used against insects and other pests. Sterilization, sex attractants, growth hormones, and feeding stimulants are used as biological pest controls. Selective, nonpersistent pesticides, herbicides, nematicides, and fungicides are being developed. Combinations of several of these approaches are providing economical and efficient integrated control of several pests.

Less attention has been given to the effect of pesticides on the nutrient composition of crops. However, it has been found that certain herbicides and soil fumigants actually step up nutrient content of the crop. For example, the use of the herbicide simazine tends to increase the protein content of small cereal grains, but usually with a loss in yield. Some herbicides and soil fumigants may also increase the vitamin content of some food crops — such as vitamin A in carrots and squash — without affecting other nutrient composition adversely.

Thus, in the development of new pesticidal agents, their impact on a food's nutrient composition should be considered. Although the changes I mentioned were increases in specific nutrients, there is no reason to believe that the effect could not be to decrease the level of an essential nutrient. Pesticides are going to have to meet increasingly stringent standards. We must keep a weather eye out for their total effect on foods.

Feed and Food Additives

In discussing food additives, we have to mention first the additives to poultry and animal feeds that may be transmitted to human food.

For example, diethylstilbestrol (DES) is used as a growth promotant for cattle. Animals legally cannot be slaughtered for meat until 7 days after they have eaten feed containing DES; but if this waiting period is not observed, or if cattle eat feed contaminated with DES during the withdrawal period, the hormone may get into the meat. Research is planned to find out how fast the hormone is depleted when used as an ear implant instead of in feed.

Our scientists have found that cooking does not appreciably reduce DES residues in meat. They believe they may be able to test cattle feces for the presence of DES instead of testing the liver after slaughter, as they do now.

Other feed additives that are withdrawn before the animals are marketed include the antibiotics used to destroy harmful organisms in swine. There is some fear that these materials might cause natural organisms common to both animals and man to develop resistance to antibiotics, and to pass this resistance on to people who eat pork. It is purely a potential problem, and further research is needed to verify or confirm this hypothesis. At Fargo, N. Dak., our scientists are studying drugs and antibiotics in livestock feed to see where the materials go . . . how they are metabolized . . . and if any residues build up.

When it comes to food additives, the meat-packing industry and its regulatory agencies are concerned now about the food additives sodium nitrite and a closely related substance, sodium nitrate, which is converted to sodium nitrite through bacterial action. One or the other is employed in practically all cured-meat products and canned meats to color and flavor them and to prevent the development of the botulism bacteria.

Recent evidence suggests that under certain circumstances, the nitrite may react with a variety of amines present naturally in many foods, drinks, tobacco smoke, and pharmaceutical drugs to form nitrosamine compounds, some of which are formidable carcinogens. Right now, the American Meat Institute, the Food and Drug Administration, and ARS scientists are cooperating in experiments to determine, once and for all, whether sodium nitrite really prevents the growth of botulism spores and, if so, what quantities are required. Preliminary evidence suggested that, at levels presently permitted in standards, the nitrite does perform an essential role in preventing botulism growth in canned meats.

At Philadelphia, our scientists have found nitrosamine at low levels in some samples of cooked sausage. The erratic occurrence of the nitrosamine suggests that some peculiar conditions in processing gave rise to its formation. We now have a comprehensive research program underway on this problem.

Other ARS scientists are analyzing some of the food additives "generally regarded as safe" to see whether chemicals added to fresh meat may mask color changes that consumers use as an index of freshness or deterioration.

CONCLUSION

In closing, I would like to emphasize that agricultural research has accomplished a great deal in protecting human health and safety — and that much remains to be done. I think that we particularly need intensified research on microorganisms and mycotoxins in our foods, on food additives, on natural toxicants in plant materials, on the essential nutrients, and on the trace elements that are indispensable to our well-being.

I would also urge you to try to get across to laymen the same attitude of objectivity regarding health scares and fads that I am sure you, as scientists and people "in the know," maintain.

For example, the magazine Fortune this month makes the point that the risks from food additives are often overstated, and that "there is no known case of any additive, used properly in a normal diet, having caused any illness other than the kind of allergenic reactions that many foods can cause." The magazine also points out that the American diet as a whole is apparently becoming safer all the time, and that our eating habits may be less likely to cause cancer than they once were.

I am confident research can develop new technology to cope with problems facing agriculture — an agriculture geared to supply growing multitudes of people with an abundance of wholesome food and materials that will continue to elevate living standards.

SESSION I: "Toxicants and Their Elimination" — Curtis R. Jackson, Georgia Agricultural Experiment Station, Experiment, Chairman

BOTULISM: A PUBLIC HEALTH MENACE?

by
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I would like to begin this presentation with a simple definition for botulism which was presented by Professor C. E. Dolman at the 1964 Botulism Symposium. He stated, "Botulism is a relatively rare, always tragic, and in some respects still mysterious, neuro-paralytic disease affecting man and animals in various parts of the world, and generally results from the ingestion of a foodstuff containing toxic metabolites from one of several known types of anaerobic bacillus, Clostridium botulinum, whose natural habitat is the soil of certain regions."

The epidemiological symptoms of botulism have been known for centuries. A classic outbreak was first reported in 1793 in Wurttemburg in Southern Germany. There was great public concern about the outbreak that involved 13 persons, of whom six died. The food vehicle suspected was "Bluze," a blood sausage prepared by filling the stomach of a pig with pork blood and "preserving" it by smoking.

In the early 1800's, Justinus Kerner, who had a unique background as mystical poet and physician, became District Medical Officer in Wurttemburg. He reported on 230 cases of sausage poisoning between 1815 and 1822. He published several monographs on the disease and attributed it to consumption of a toxic fatty acid which formed in the sausage during storage. Kerner's work led to the public health regulation that sausage poisoning had to become a reportable disease in 1824!

The classical case of botulism occurred in Ellezelles, Belgium, in 1895 when an outbreak took place among 34 members of a musical society. The musicians participated in a post-funeral meal that included raw, salted ham. Thirteen of the 34 developed the neuro-paralytic syndrome of botulism within 20 to 36 hours after the meal, and three of the 13 died. Van Ermengem of Ghent isolated a spore-forming bacillus from a remnant of the implicated ham, as well as from the spleen of one of the victims. The culture filtrates of the microorganism produced characteristic fatal neurological syndromes when injected into various species of laboratory animals. He contended that a metabolite was produced within the ham as the organism grew in it. Van Ermengem drew the parallel between the Ellezelles fatalities to the sausage poisoning cases described by Kerner, for which Muller had coined the term "botulism" in 1870. Van Ermengem then proposed that the causative microorganism be called Bacillus botulinus after the Latin term for sausage.

Van Ermengem did a remarkable job in the study of this microorganism and reported on the following phenomena:

(1) He recognized that the disease could be caused by different types of sausages and preserved meats — but also by fishery products.
(2) He stated that the "fish-poisoning" reported in Russia was apparently identical with botulism and could be distinguished from shellfish poisoning.
(3) He considered the organism a saprophyte that produced no toxin in the living animal.
(4) He calculated that botulinum toxin is the

most potent toxin produced by the bacteria.

(5) Marked differences in susceptibility of various laboratory animal species were also noted to the administration of toxic ham extracts or culture filtrates.

(6) The toxin was reported to be sensitive to light, air, alkalinity, and heat
30 minutes at 80° C. = almost completely
or 1 hour at 70° C. inactivated the toxin
5 minutes of boiling completely destroyed activity

(7) The toxin proved resistant to acidification, to precipitation by alcohol and ammonium sulfate, to dialysis, and to proteolysis.

(8) Best growth of the organism was reported in a chopped pork medium containing 1 percent glucose, 1 percent peptone, 1 percent sodium chloride, and 2 percent gelatin between 20° to 30° C. Poorer growth was obtained when the salt concentration was increased to 5 percent, but growth was reduced at higher salt concentrations. Cultures liquified gelatin slowly.

Van Ermengem's excellent description of the properties of this anaerobic bacillus indicate that we would classify this as a non-proteolytic type B strain. This first outbreak of human food poisoning caused by this toxin type in North America did not take place until 1958 among an Indian tribe in Northern British Columbia.

Van Ermengem's strain of C. botulinum, and indeed all other strains, may be characterized by the unique and specific toxin they produce. The group of C. botulinum microorganisms can be divided into 6 types, designated A through F, based upon the immune response that the antigenic toxin produces in animals. The type C is somewhat unique in that it can be divided into two sub-groups, C_a and C_b. In most cases for the research worker, clinician, or public health epidemiologist, the cultural characteristics of the strains producing the toxin may only be of secondary interest. Thus, a strain of an anaerobic sporeforming bacillus that produces a toxin antigenically similar to another botulinum toxin is a strain of C. botulinum irrespective of what other kinds of characteristics it exhibits. There have been recent thorough studies that examined the cultural and biochemical characteristics that C. botulinum possesses. These studies by Holdeman and Smith not only included a wide variety of strains of botulinum, but culturally similar other clostridia were also included.

The results of some selected reactions for the proteolytic strains of botulinal types A, B, and F and those of C. sporogenes are presented in table 1. There are no cultural or morphological characteristics that can be used to reliably separate the botulinum types from the non-toxic C. sporogenes. The data for the type F botulinum include that for the Langeland and the Argentine strains.

Table 2 presents data for the saccharolytic botulinum types D and C and Clostridium novyi. The C. novyi is toxinogenic as well, and also produces a gas gangrene in man and animals and possesses a lethal toxin plus a lecithinase.

Table 1. Reactions of proteolytic strains of C. botulinum types A, B, and F and Clostridium sporogenes (Group 1)

Characteristic	Botulinum			C sporogenes
	Type A	Type B	Type F	
Toxicity	+	+	+	–
Proteolytic (digests meat, gelatin, or milk)	+	+	+	+
pH in glucose medium	5.4 to 6.0	5.8 to 6.1	5.9	5.5 to 6.2
Acid produced from:				
glucose	+	+	+	+
fructose	+	–	–	–
d-maltose	–	–	–	–
sucrose	–	–	–	–
demannose	–	–	–	–
H_2S production	+	+	+	+
Hydrolysis of esculin	+	+	+	+
Growth in 6.5 pct. NaCl	±	+	–	+
Hemolysis (Equine RBC)	+	+	+	+
Lipase	+	+	+	+

Table 2. Reactions of Saccharolytic C. botulinum types D and C and Clostridium novyl

	Botulinum		
Characteristic	Type D	Type C	C. novyl
Toxicity	+	+	+
Digests:			
gelatin	+	+	+
milk	+	−	−
pH in glucose medium	5.2	5.2 to 5.7	5.5 to 5.6
Acid produced from:			
glucose	+	+	+
fructose	−	±	−
d-maltose	−	±	+
sucrose	−	−	−
d-mannose	−	+	±
H_2S production	−	−	−
Hydrolysis of esculin	−	−	−
Growth in 6.5 pct. NaCl	−	−	−
Hemolysis	+	+	+
Lipase	+	+	+
Lecithinase	−	−	+

Table 3 presents data saccharoytic botulinum types E, B, and F and includes the "E-like" clostridia that are culturally and serologically identical to the type E strains but do not produce toxin. The other strains, which are serologically related but not culturally somewhat different, have not been included. The only other clostridia similar to these in the third group are Clostridium chauvoei and C. butyricum but these are lactose positive, while all of the C. botulinum types do ferment lactose. Thus, the "E-like" group is a unique group which can be considered to be nontoxic variants of type E C. botulinum.

The major physiological end products of the growth of each of these three groups of microorganisms are presented in table 4. The principal organic acids produced by the proteolytic group are acetic, butyric, isobutyric, and isovaleric acids. Spermine and spermidine are the amines produced in greatest amount by this group. The first saccharolytic group produces chiefly pro-

Table 3. Reactions of saccharolytic C. botulinum types E, B, and F and non-toxic "C. botulinum type E"

	Botulinum			
Characteristic	Type E	Type B	Type F	"E-like"
Toxicity	+	+	+	−
Digests:				
gelatin	+	+	+	+
milk	−	−	−	−
pH in glucose medium	5.3 to 5.6	5.0 to 5.4	5.0 to 5.5	5.0 to 5.5
Acid produced from:				
glucose	+	+	+	+
fructose	+	+	+	+
d-maltose	±	+	+	+
sucrose	+	+	+	+
d-mannose	+	+	+	+
H_2S production	−	−	−	−
Hydrolysis of esculin	−	−	−	−
Growth in 6.5 pct. NaCl	−	−	−	−
Hemolysis	+	+	+	+
Lipase	+	+	+	+
Lecithinase	−	−	−	−

Table 4. **Major products of C. botulinum and related Clostridia resulting from glucose in chopped meat medium**

Proteolytic Botulinum A, B, F C. sporogenes		Saccharolytic Botulinum C, D C. novyl		Botulinum E, B, F and "E-like"	
ACETIC	3.0-4.0[1]	ACETIC	0.0-1.0	ACETIC	1.0-1.5
propionic	0.2-0.3	PROPIONIC	1.5-2.5		
ISOBUTYRIC					
BUTYRIC					
ISOVALERIC	3.0-4.0	BUTYRIC	1.5-3.0	BUTYRIC	1.5-2.0
Valeric		valeric			
isocaproic					
lactic	trace	lactic	tr-mod	lactic	tr-mod
succinic	trace	succinic	tr-mod	succinic	tr-mod
		formic	trace	formic	tr-mod
alcohols	moderate	alcohols	trace	alcohols	trace
spermine	moderate	isoamylamine-moderate		trace	
spermidine	moderate	phenethylamine-moderate		of all 4 amines	

[1] Milliequivalents per 100 ml.

MAJOR AMOUNT	< 1.0 meq./100 ml.
MODERATE AMOUNT	0.25-1.0 meq./ml.
minor amount	< 0.25 meq./ml.

pionic, butyric, and acetic acids and the amines isoamylamine and phenethylamine. Only acetic and butyric acids are produced in considerable quantities in the second saccharolytic group while a mixture of all four amines are produced in reduced quantities by this group.

These physiological data thus indicate that there are marked physiological differences among C. botulinum types with stronger physiological relationships to other clostridia within any one group than between C. botulinum types in all three groups.

Let us now direct our attention to the frequency of the disease syndrome of botulism. Since 1895, more than 6,000 cases of botulism have been reported in the world. More than 1,700 deaths among the 6,000 cases resulted in an overall mortality rate of about 30 percent. If we compare 1,700 fatalities with the more than 1,500 published materials on botulism, we obtain about one publication for every fatality!

There should be little disagreement that the disease has elicited worldwide interest.

In the United States, considerable interest in the disease has been shown since World War I when the U.S. Department of Agriculture encouraged consumers to preserve more food. They also recommended that the "cold-pack" method be used to process these foods in jars or cans and the net result is shown in figure 1.

The relationship of home-canned foods to the total outbreaks of botulism are shown in figure 2. The relatively minor contribution of commercially canned foods to the total of botulism outbreaks — with the exception of the 1920's and 1963 — is also shown in this figure.

The foods involved in botulism outbreaks in the United States and Canada are shown in figure 3. The commercially canned products made up 10 percent of the total outbreaks. Overall mortality was about 65 percent. A general trend of major involvement due to vegetables with less involvement due to meat products is evident.

The types of C. botulinum that most frequently cause human botulism, are presented in table 5 and in figure 4.

Interesting data on the average mortality rate for botulism from the 1900's to the present are presented in figure 5. The average mortality has decreased from about 60 percent from 1900 to 1945 to about 25 percent at present.

The ability of specific foods to support growth and toxin production by type E C. botulinum can be deduced somewhat inferentially by examining the foodstuffs involved in type E botulism outbreaks (table 6).

Type E C. botulinum has not yet been isolated from meat products most probably because of relatively low incidence of all types of C. botulinum spores in foods. Reports by Taclindo and others (1967) found only one positive type B sample in 73 samples of luncheon meat they examined, and of a total of 113 samples of 64 convenience foods, only raw unshucked oysters were found to contain type E spores. When the 64 convenience foods were inoculated with type E spores only turkey roll and soybean cake were found to contain toxin. However, the other 60 food products did contain viable type E spores. These data indicate limited toxin-producing ability of the organism in most foods but a general ability for the spores to survive in these products. Studies by workers at General Foods found that only one sample of 400 convenience foods (boil-in bag, vacuum-packaged, pressurized, and dehydrated or freeze-dried)—in this case vacuum packaged frankfurters—was found to contain type B spores.

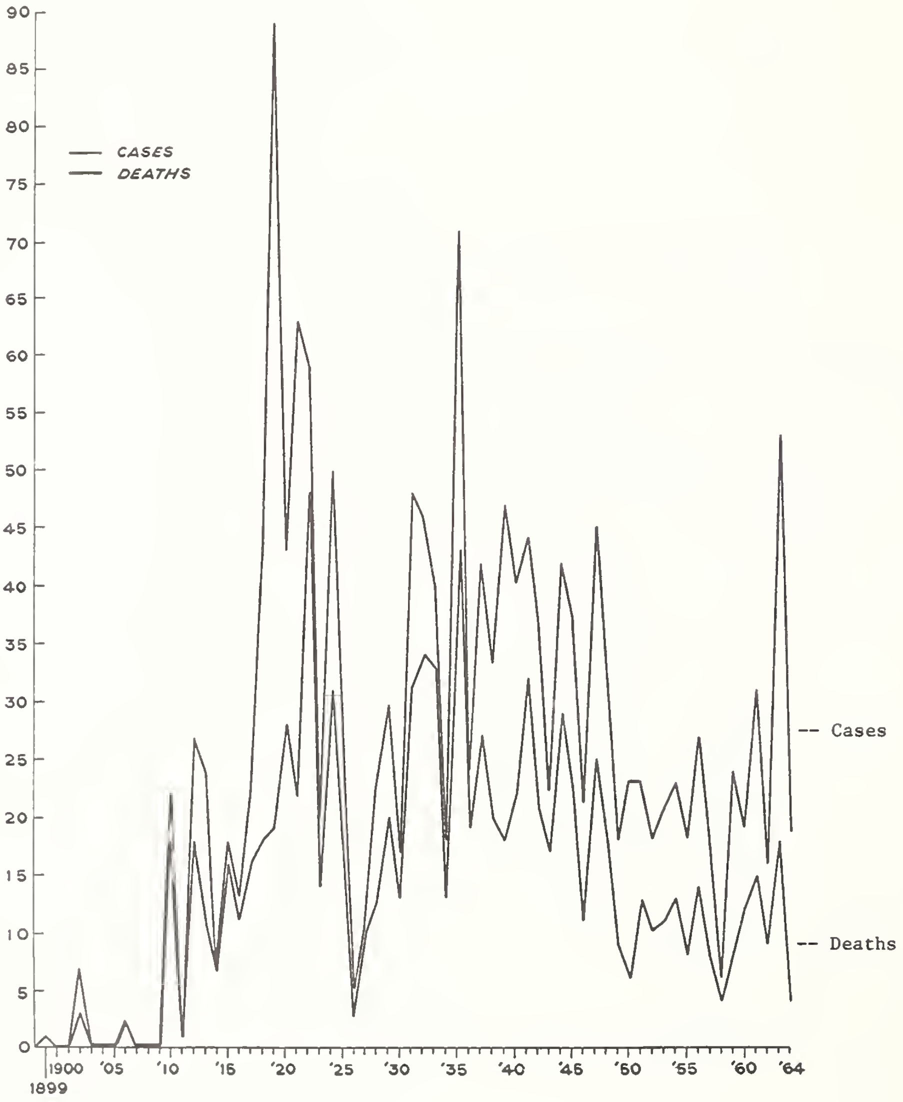

Figure 1. Sixty-five years of botulism in the United States and Canada

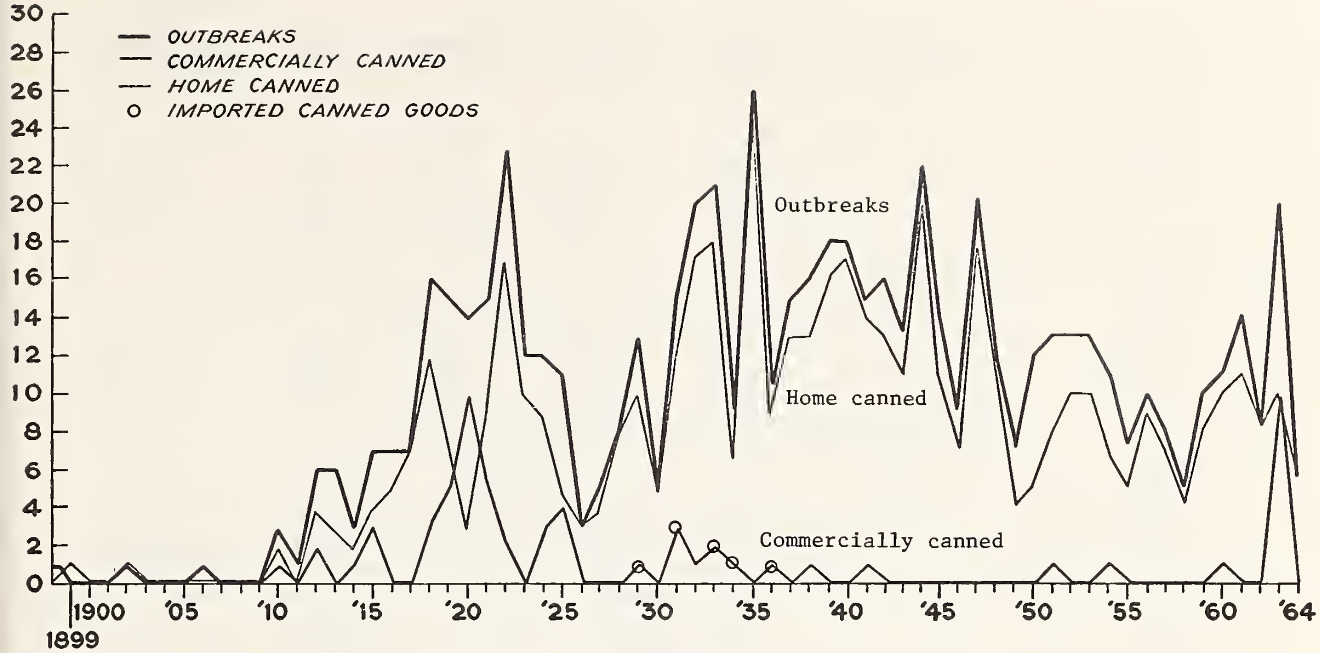

Figure 2. Sixty-five years of botulism in the United States and Canada

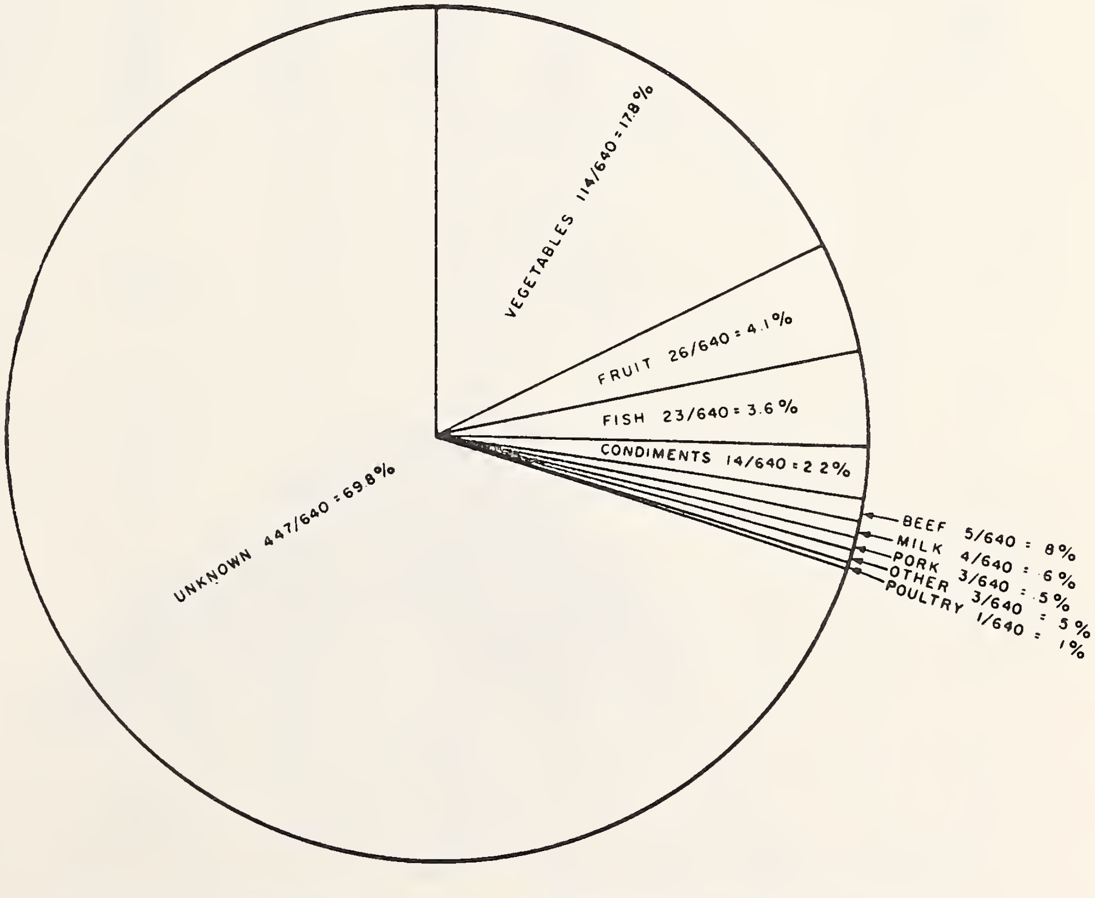

Figure 3. Foods involved in botulism outbreaks in the United States 1899-1967

Table 5. Botulism cases and deaths by toxin types for various periods

Toxin types	1930-39	1940-49	1950-59	1960-67	Total 1899-1967
Type A					
Deaths	69	62	18	4	278
Cases	94	110	39	20	463
Mortality	73.4 pct.	56.4 pct.	46.2 pct.	20.0 pct.	60.0 pct.
Type B					
Deaths	16	12	2	2	59
Cases	33	22	4	19	121
Mortality	48.5 pct.	54.5 pct.	50.0 pct.	10.5 pct.	48.7 pct.
Type E					
Deaths	2	1	7	15	25
Cases	6	3	14	34	57
Mortality	33.3	33.3	50.0	44.1	43.9
TYPE F					
Deaths				0	0
Cases	–	–	–	3	3
Mortality				0 pct.	0 pct.
Subtotal, unknown toxin types					
Deaths	162	119	58	12	584
Cases	245	181	176	79	1019
Mortality	66.1 pct.	65.7 pct.	32.9 pct.	15.2 pct.	57.4 pct.

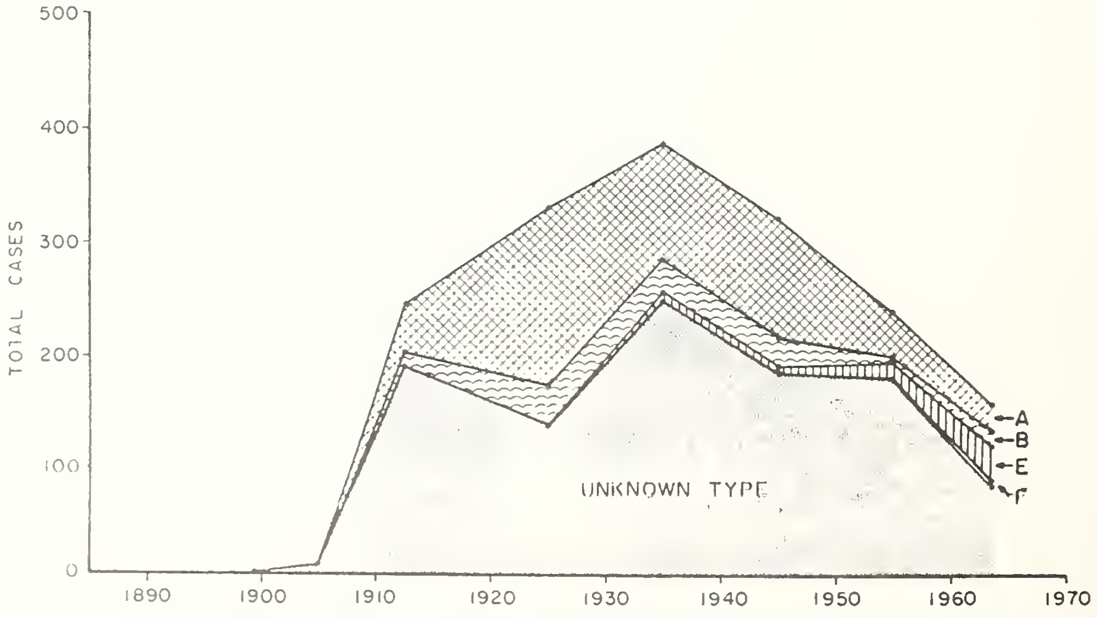

Figure 4. Cases of botulism, by type in 10-year periods 1899-1967

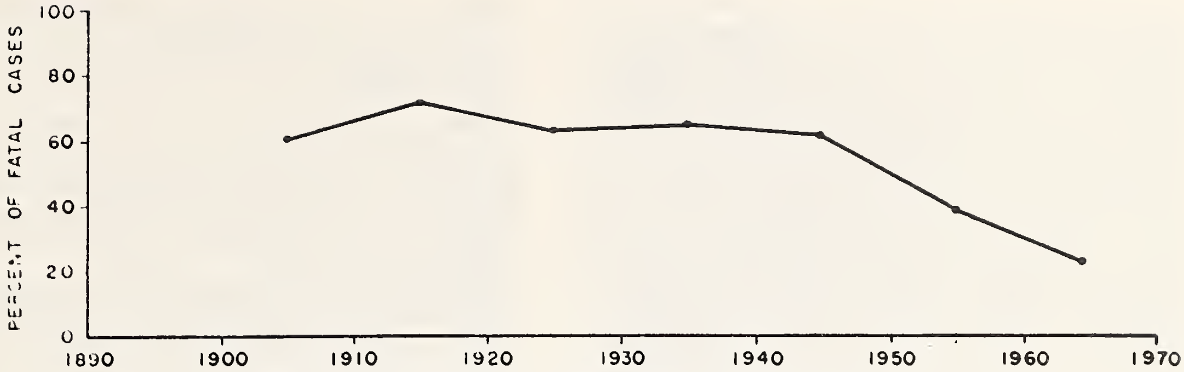

Figure 5. Botulism death-to-case ratios by 10-year periods

Table 6. Type E botulism in the United States and Canada

Year	Location	Preparation	Food	Outbreaks	Cases	Deaths
1932	New York	C.C.	Smoked salmon	1	3	1
1934	do	C.C.	Sprats	1	3	1
1940	Canada	H.C.	Salmon eggs	1	5	3
1941	California	C.C.	Mushroom sauce	1	3	1
1944	Canada	H.C.	Salmon	1	3	3
1949	do	H.C.	Herring, pickled	1	2	1
1950	Alaska	H.P.	Beluga flippers	1	5	0
1952	do	H.P.	Beluga flippers	1	1	1
	Canada	H.P.	Trout	1	1	1
1954	do	H.P.	Salmon egg cheese	1	3	1
1956	Alaska	H.P.	Beluga flippers	1	2	1
	Canada	H.P.	Seal flippers, uncooked	1	8	6
1957	do	H.C.	Salmon eggs	1	3	3
1959	Alaska	H.P.	Grey whale fluke	1	7	1
	do	H.P.	Salmon eggs	1	1	1
1960	do	H.P.	Salmon egg cheese	1	2	2
	Canada	H.P.	Salted Holland herring	1	1	0
	Minnesota	C.C.	White fish, smoked (vacuum packed)	1	2	2
1961	Canada	H.P.	Fish eggs	1	4	0
	do	H.P.	Fish eggs	1	3	2
	Washington	H.C.	Salmon eggs, uncooked	1	4	1
1962	Canada	H.P.	Seal flippers	1	1	1
	do	H.P.	Salt meat, fermented	1	1	1
1963	Alabama	C.C.	Smoked whitefish (ciscoes)	1	1	0
	Alabama	C.C.	Smoked whitefish	1	2	0
	Canada	H.P.	Salmon eggs	1	2	1
	Kentucky	C.C.	Smoked ciscoes	1	2	0
	Michigan	C.C.	smoked ciscoes	1	2	2
	do	C.C.	Tuna fish	1	3	2
	Minnesota	C.C.	Smoked ciscoes	1	1	0
	Tennessee	C.C.	Smoked ciscoes	1	7	2
	do	C.C.	Smoked ciscoes	1	5	3
1967	Alaska	H.C.	Seal flippers	1	1	0
1968	do	H.C.	Fish in seal oil	1	1	0
Totals				34	95	44

The fluorescent antibody technique has proved useful in detecting the presence of C. botulinum microorganisms in sample material as well as in confirming the type of various isolates detected. Russian workers including Kalitina (1960) and Bulatova and Kabanova (1960) were among the first to find this technique useful for identifying types of C. botulinum when they observed that certain types of luminescent sera would react only with certain types of C. botulinum. Batty and Walker (1963) and Walker and Batty (1964) were able to separate C. botulinum into three groups using fluorescent antibody staining: (a)proteolytic types A, B, and F; (b) nonproteolytic type E and D; and (c) nonproteolytic type E. Boothroyd and Georgala (1964) also found that fluorescent antibody staining showed considerable promise as a tool for isolation or identification of C. botulinum or both. These same workers (Georgala and Boothroyd, 1967) later indicated that more work was required on the serological characteristics of cells, spores, and flagella of each type before the technique can be widely accepted. Walker and Batty (1967) further observed that the fluorescent antibody technique could be used in the identification of toxic as well as nontoxic strains of C. botulinum. Ward, Carol, and Garrett (1967), however, were unable to obtain consistent results with respect to toxin production and fluorescence when mouse-toxin tests were compared with fluorescent detection of toxin.

Hobbs and Anderson (1967) used polyacrylamide gel electrophoresis to study relationships between C. botulinum and nontoxic clostridia or nontoxin mutants. They observed that protein patterns of vegetative cell extracts of all strains of type E examined were essentially the same and that each of the types A,B, and C gave different characteristic patterns.

Midura and others (1967) examined the possibility of using immunofluorescence for detection of C. botulinum types A, B, and E. They reported that this technique could be quite useful as a presumptive indicator of the presence of C. botulinum cells. Midura and others (1968) further combined the use of this technique with animal tests to detect growth and toxin production by C. botulinum type E in experimentally inoculated packages of turkey rolls. They were able to obtain a time relationship between the presence of vegetative cells, determined by immunofluorescence, and the presence of toxin as demonstrated by animal tests.

Immunological techniques have also been reported to be useful in detecting toxins of C. botulinum. Rycaj (1956), Yafayer and Chepelir (1961), and Sinitsyn (1961) indicated that passive hemagglutination of botulinal antitoxin-coated, tanned sheep red blood cells was as sensitive in detecting botulinal toxins as animal assays. Johnson and others (1966) were able to detect one LD_{50} mouse unit of botulinal toxin using hemagglutination. Vermilyea, Walker, and Ayres (1968) have employed the Ouchterlony immunodiffusion technique to detect toxins of C. botulinum. They indicated, however, that the mouse assay was a more sensitive method for detecting toxin since the toxin had to be concentrated with Sephadex when the gel diffusion technique is used.

More recently, Duda and Slack (1969) have demonstrated toxin production by C. botulinum type A using electron microscopy. Upon electron microscopic examination of ultrathin sections of spheroplasts of the organism which had been exposed to ferritin-labeled type A antitoxin, they observed the presence of ferritin labeled antibody in the outer spore coats and in vegetative cell cytoplasm. No ferritin-labeled antibody was observed in the spore cortex or spore core.

Reiner (1965) was the first to investigate the feasibility of using PGLC for the identification of bacteria. Upon pyrolysis of cells of E. coli (18 different antigenic strains), Shegalla sp., group A Streptococcus pyogenes (4 types), and mycobacteria (10 different pathogenic and forms) each strain produced its own unique pyrogram and different cultures of the same strain yielded similar profiles. Reiner (1967) in a further study using additional microorganisms which were somewhat difficult to classify, reconfirmed his earlier report that PGLC could be used in the classification of microorganisms. Reiner and Ewing (1968) further observed that this technique could be used to differentiate two cultures of E. coli which differed only in their flagellar, or H, antigens. These antigens were composed almost entirely of protein, thus, indicating that slight protein differences have a strong influence on pyrograms obtained.

Previous mention has been of the relatively low incidence of botulism in the world since its discovery. The hazard due to botulism in the United States should thus be placed in its proper perspective. During the past 45 years in the United States, an average of more than 29 billion cans of food have been processed each year. A total of 775 billion cans of food were thus commercially processed over this 45 year period and during this time there have been only three episodes of botulism due to consumption of commercially processed canned food. One outbreak occurred in 1971 in California where the death of one person took place after canned mushrooms were eaten. Two deaths in 1963 were the result of consumption of one can of tuna processed in California but consumed in Michigan. One can of vichyssoise produced the death of one person in 1971 in New York, so we thus have three outbreaks of botulism food poisoning that resulted in the deaths of four people out of the total of 775 billion cans of food that were processed by commercial firms.

Home canned foods remain a much greater risk to the consumer since an average of five to ten outbreaks are produced each year that involve 10 to 20 people and result in from two to five deaths caused by eating these products. If one considers the relatively low numbers of cans or jars of food processed in homes, the probability of getting botulism from home canned foods are considerably greater than the 100 billion to 1 odds against getting botulism from commercially canned foods.

Therefore, any swollen or bulging cans or bulging ends of jars of food should never be opened and the contents tasted. All home canned food should be thoroughly boiled for 20 minutes before serving and should be immediately served after heating to inactivate any toxin that may be present. This procedure and the strict adherence to all U.S. Department of Agriculture recommendations on the proper home preparation of canned food should serve to increase the safety of these products.

MYCOTOXINS

by
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Mycotoxins have been responsible for human intoxications for thousands of years. Mushroom poisoning was recorded before the fifth century B.C. and ergotism or Saint Anthony's Fire—as it was called then—was traced to the ingestion of infected rye and other cereals in the Middle Ages (Rothlin and Cerletti, 1954) and was reported to be responsible for several epidemics. Outbreaks of a mycotoxicosis, alimentary toxic aleukia (ATA) were recorded in Russia as early as the 19th century, and the toxicity of smut and rust fungi for man and animals was actively investigated in that country during the second half of the 19th century (Bilay, 1960). Late in the 19th century, molds were recognized in Japan (Sakaki,1891) as the agents responsible for neurotoxic effects in animals. At that same time, Sakaki advanced the opinion that beriberi was caused by the consumption of moldy rice but did not identify the fungus or its toxin. Even today, Uraguchi (see Saito et. al. 1971) is convinced that Shoshinkakki (acute cardiac ceriberi) must be considered a mycotoxicosis.

In the United States, Butler (1902) produced leucoencephalitis in a horse by feeding it moldy ear corn. Alsberg and Black in 1913 concluded that products of mold growth could be involved in disease and Schofield (1924) was among the first in this country to associate mold-infected feeds with toxicity to domestic animals. Despite these early studies relating mold-contaminated foods and feeds with disease, it was not until 1943-44 that the Russians observed and definitively related a severe and widespread epidemic of man and animals in the Orenburg district of Russia to the consumption of moldy millet (Joffe, 1963). The death of large numbers of horses throughout the district were recorded and the development in man of ATA or septic angina on an epidemic scale was noted among those consuming grain that had remained in fields that had been cut but not harvested during the siege of Stalingrad in the winter of 1942-43. Clinical manifestation of the disease include leukemia, agranulocytosis, necrotic angina, hemorrhagic diathesis, sepsis, and aregenerative exhaustion of the bone marrow.

Numerous isolates from the genera Fusarium, Cladosporium, Alternaria, Penicillium, and Mucor were associated with the exposed grain (Joffe and Mironov, 1947; Stukov and Mironov, 1947; Myasnikov, 1948; Joffe, 1950), but it was not until later that Joffe (1960, 1962) and Bilay (1960) reported on the prevalence of Fusarium mycotoxicosis in diseased grain and pointed out the psychrophilic character (i.e. growth at -2° to -10° C.) and other biological properties of these fungi.

Similarly in 1940 (I. Miyake et. al., 1940; see Saito et al., 1971) Japanese investigators observed that a fungus found on domestic and imported rice caused yellowing of the grain which they associated with nervous disorders. A possible toxic effect was reported when rats were fed the moldy rice. Shortly after World War II, Tsunoda (1948; 1951; 1952; 1953 a,b) indicated that Penicillium islandicium Sopp and several other penicillia produced hepatic lesions and other serious liver damage in experimental animals.

In 1953, Sippel et al., described a disease of pigs and cattle that resulted when those animals had consumed moldy corn. Forgacs and Carll (1955) suggested that hemorrhages in many of the tissues of broilers might be a moldy feed toxicosis and Burnside et al. (1957) showed that corn infected with Penicillium rubrum was toxic to pigs, mice, and horses. Later, Forgacs et al. (1958, 1962) demonstrated that when several Penicillium and Aspergillus isolates were cultured in poultry feed, the feed was capable of inducing hemorrhages and congestion of the muscles and several of the vital organs of chickens.

In addition to these significant, yet isolated and often cursory observations by scientists in various parts of the world, microbiologists began a concerted intensive and systematic search in the mid-1940's of the antibiotic producing potential of selected species and strains of Penicillium, Aspergillus, and Fusarium. While many of these studies yielded substances that were therapeutically useful, investigations generally were abruptly halted when the mold metabolite was found to be toxic.

In 1960, two separate but highly significant events were reported to the scientific community. One of these involved the loss of turkey poults fed moldy peanut meal. For lack of knowledge at that time of its true cause, the disorder was called "Turkey X Disease" by workers at the Tropical Products Institute in Britain (Blount, 1961). Brazilian groundnut (peanut) meal was identified as the source of the toxic agent. Shortly thereafter, it was found that the disease resulted from groundnuts processed in East Africa as well as those in Brazil and that the toxicity was not limited to turkeys — severe losses of ducklings from a similar disease occurred in Kenya and Uganda (Asplin and Carnaghan, 1961). Poisoning in ducklings was characterized by loss of appetite, lethargy, and wing weakness. Liver parenchyma cells of young birds that had ingested the suspect meal for a few days were necrotized. There was excessive proliferation of bile duct cells (Sargeant et al., 1961 a,c). Examination of the feed revealed that the mold Aspergillus flavus Link ex Fries produced the toxin (Sargeant et al., 1961 c) and that a characteristic blue fluorescence was observed in the toxic meal. Further work soon demonstrated that the toxic material was extractable with chloroform and consisted of four fractions which were separable by chromatography and fluoresced as individual spots when exposed to long-wave ultraviolet light (Nesbitt et al., 1962).

At about the same time (1960) hepatomas of hatchery-reared rainbow trout, Salmo gairdneri, appeared in epidemic proportions at various localities in the United States and Europe (Halver and Mitchell, 1967). The appearance in private, State, and Federal hatcheries of trout hepatoma followed the introduction of dry, pelleted commercial feeds as a primary dietary source. Hepatomas were also observed in brook trout (Salvelinus fontinalis) in cutthroat trout (Salmo clarki), and in brown trout (Salmo trutta) (Dawe et al., 1964). When known hepatomagenic diets were fractionated, four lipid soluble carcinogenic agents were isolated (Sargeant et al., 1961 c).

AFLATOXIN

After the isolation of toxic material by workers at the Tropical Products Institute in London and the Central Veterinary Laboratory in Weybridge, it was quickly learned that certain strains of the common mold Aspergillus flavus that grew in damp peanut meal or cottonseed products were able to synthesize highly substituted coumarins containing bifuran rings and lactone configurations (Asao et al., 1963, 1965). Owing to their blue or green fluorescence when exposed to ultraviolet light, these components were designated as aflatoxins B_1, B_2, and G_1, G_2, respectively. Allcroft and Carnaghan (1963) reported two hydroxy-aflatoxin derivatives, M_1 and M_2 were in the milk of animals eating feed experimentally contaminated by toxic groundnut meal. Later M_1 and M_2 were isolated from several samples of market milk in South Africa (Purchase and Vorster, 1968). Two additional hydroxy derivatives of aflatoxin were described by Dutton and Heathcote (1966, 1968) and have been designated as aflatoxins B_{2a} and G_{2a}.

In 1971, Dalezios et al., (1971) isolated a phenolic derivative of aflatoxin B_1 appearing mainly in conjugated form in the urine of rhesus monkeys. Since the metabolite is a phenolic product, they suggested that it be called aflatoxin P_1. They further proposed that since P_1 represents at least 60 percent of the urinary aflatoxin derivatives, its identification in human urine might facilitate estimation of aflatoxin exposure in human populations.

Certain strains of the Aspergillus flavus group have definitely been shown to produce aflatoxin; for example, Aspergillus flavus Link ex Fries, and A. parasiticus Spear (Skau, 1965). While a number of other strains have been implicated (Hodges et al., 1964; Kulik and Holady, 1967; Basappa et al., 1967; Scott et al., 1967; Walbeek et al., 1968), several workers have failed to duplicate these findings (Wilson et al., 1967 a, 1968; Mislivec et al., 1968; Hesseltine et al., 1966). In addition to the aflatoxins mentioned above, several more remotely related carcinogenic compounds including two sterigmatocystins and, more recently (Rodricks et al., 1968, a,b) aspertoxin have been associated with this group and with A. versicolor and A. nidulans as well (Holzapfel et al., 1966). Sterigmatocystin has been postulated to be a precursor of the aflatoxins, and aspertoxin is a derivative of sterigmatocystin (Rodricks et al., 1968 a,b). Within this family of mold metabolites, B_1 is the most acutely toxic with an LD_{50} of 0.36 mg./kg. M_1 is almost as active followed by G_1 (0.79 mg./kg.), B_2 (1.8 mg./kg.), M_2 and

G_2 (3.45 mg./kg.). Aspertoxin is only 1/25 toxic as B_1 and the toxicities of B_{2a}, G_{2a} and sterigmatocystin are 1/60, 1/100, and 1/250 that of B_1, respectively.

In tropical countries, climatic conditions and primitive cultural practices provide favorable conditions for contamination of foodstuffs by fungi. Much of the damage sustained by peanuts occurs after the crop has been dug but before it is dry. Mechanical damage in harvesting, improper drying and storage, and insect infestation are important factors (Ashworth and Langley, 1964). Indian workers (Parpia and Sreenivasamurthy, 1970) reported that practices such as manual harvesting of peanuts after flooding fields with water to soften the earth, storing crops when the commodity is still wet or, if dry, storing in underground pits with poor protection from water seepage, and harvesting in humid weather tend to raise the moisture level of crops and provide conditions that favor mold development.

Experimentally, a wide variety of commodities have been shown to yield aflatoxin in the presence of suitable strains of A. flavus but the presence of these toxins in naturally contaminated crops is much less certain. However, aflatoxins have been detected in: barley, bean, brazil nut, cassava, cocoa bean, cocoa product, coconut oil cake, copra, corn, corn grits, cottonseed meal, cowpea, locust bean, millet, palm kernel, pea, peanut, peanut meal, peanut butter, pepper--black, raisin, rice, sesame, sorghum, soybean meal, wheat, and wheat flour.

The discovery that aflatoxins present in moldy feeds were responsible for liver damage in many species of fish, birds, and mammals and the demonstration that these toxins induce tumors in ducklings, rats, trout, and monkeys has caused world wide concern. Not only have the aflatoxins been proved to be carcinogenic (Neuhaus and Halver, 1968) and teratogenic (Di Paolo et al., 1967; Elis and Di Paolo, 1967) for various laboratory animals, but Newberne et al., (1966) have shown that aflatoxin was responsible for hepatitis in dogs and Wilson et al., (1967 b) have more broadly implicated aflatoxins in a number of epizootics of farm animals in many Southern States.

While the danger that these toxic compounds present to lower animals has been amply demonstrated, their full carcinogenic potential for man remains unknown. Evidence continues to mount implicating aflatoxins as human carcinogens. An article in Journal American Medical Association (Anonymous 1963) speculated on the relation of moldy diet components to the high incidence of hepatoma among African Bantu tribes. Among the Bantu, hepatomas represent 68 percent of carcinomas and the rate for primary hepatomas of 14 per 100,000 population contrasts markedly with 3.2 for U.S. negroes, 1.7 for U.S. whites, and 0.18 for Danes. Also in 1963, Schoental(1963) and Davidson (1963) pointed out that cirrhosis and liver carcinoma may be caused by fungal toxins. Later, data by Dunham and Bailer (1968) indicated that more than half of all carcinomas in Southern Africa are liver tumors — an incidence 5 to 50 times higher than that reported in the United States.

Coady (1965) obtained limited clinical data suggesting the possibility of an etiological relationship of aflatoxin to human disease and several investigators have related the increased incidence of liver tumors to the ingestion of hepatotoxic fungal products (Carnaghan and Crawford, 1964; Kraybill and Shimkin, 1964; Oettlé, 1964; Isaacson, 1966; and Diouf, 1967). Oettlé (1964) reported extensive liver cancer in Japan and other Asiatic countries where customary dietary patterns include sundry mold-fermented foods. Alpert et al., (1968) have related regional high incidence of hepatomas to the necessity of ingesting contaminated foods because of food scarcity.

More direct evidence was reported by Payet et al., (1966), Serck-Hanssen (1970), Parpia and Sreenivasamurthy (1970, 1971), and Bosenberg and Morgenroth (1972). The first reported incident (Payet et al., 1966) involved two African infants who when less than a year old, unknowingly, consumed protein concentrates that contained 10 mg. aflatoxin/kg. of feed. Liver biopsies made 4 years later indicated alterations of parenchymal cells. For one of these, changes were not considered pathological but for the other they were adjudged to be precirrhotic. After an additional 2 years, the one child had inflammatory fibrotic liver lesions but carcinoma was still not observed.

Serck-Hanssen (1970) provided a case report of an acute hepatic disease in a 15-year-old Ugandan boy strongly suggesting that ingestion of aflatoxin contributed to the boy's death. The aflatoxin-induced fatal hepatitis resembled that seen in African monkeys poisoned by aflatoxin (Alpert et al., 1970). Moldy cassava constituted the youngster's staple diet and analysis of a specimen from the stock indicated an unusually high concentration of aflatoxin, that is, 1.7 mg./kg.

Parpia and Sreenivasamurthy (1970, 1971) reported in 1969 that during treatment for kwashiorkor, three of 20 children who accidentally consumed some commercial peanut meal having an aflatoxin content of 300 ug/kg. of meal, developed hepatomegaly after 3 weeks. Needle biopsies from the livers of 17 more of the group receiving this meal for a period varying from 1 week to one month showed pathologies progressing from fatty liver infiltration to different grades of 'Indian childhood cirrhosis' within a period of a year.

Recently, Bosenberg and Morgenroth (personal communication 1972) found an acute yellow liver atrophy from a post-mortem sectioning of a man who had consumed extraordinary amounts of various nuts. The patient suffered an iron imbalance which was expressed in a liver cirrhosis that alone was considered too superficial to be responsible for death.

The first report of the detection of aflatoxins in human tissues was made by Shank et al. (1971). These workers detected as much as 93 ug. B_1/kg. in a liver specimen, 123 ug./kg. in stool, 127 ug./kg. in stomach and intestinal contents and 8 ug./ml. in bile. In addition, trace amounts were detected in brain, kidney, and urine. Further, they indicated that several hundred children in Thailand between the ages of 1 and 13 years die of disease of unknown etiology but of cases seen in hospitals, the disease was manifested by vomiting, convulsions, and coma resulting in death (80 percent). Chemical analysis of autopsy specimens from 23 Thai children who died with acute encephalopathy and fatty degeneration of the viscera (EFDV) revealed the presence of aflatoxin B_1 in one or more specimens from 22 of the 23 EFDV cases. Further, Olson et al. (1971) reported that the extent of aflatoxin contamination in food samples from Thai markets seemed to parallel this acute disease both seasonally and geographically. In addition, Bourgeois et al. (1971) drew attention to the marked similarities between Reye's syndrome (acute aflatoxin B_1 poisoning in young macaques) and EFDV in Thai children and suggested that aflatoxins and possible other mycotoxins may be a factor in the etiology of EFDV.

The age of the animal is of great importance in determining its susceptibility to aflatoxin. Almost invariably, the young of the species is more sensitive than the mature animal. Day-old ducklings were found to be especially susceptible and were employed by Asplin and Carnaghan (1961) in the initial development of a biological assay procedure. The single oral LD_{50} dose for male ducklings was reported by Butler (1964a) to be 0.4 mg./kg. compared with 7.2 mg./kg. for male rats. However, Carnaghan (1965) found the male Fischer rat and the duck to have similar susceptibility (0.03 p.p.m.). In 1969, Halver showed that trout fry were even more sensitive to this metabolite (0.002 p.p.m.). With each 4 week increment of time preceding a first feeding of aflatoxin, the incidence of hepatoma decreased. In fish held for 20 weeks before being challenged with aflatoxin, tumor incidence was 11 percent that of a population containing this toxin in the dietary throughout the interim.

Allcroft (1969) indicated that with poultry the order of susceptibility to aflatoxicosis was: ducklings $\wedge$ turkey poults $\wedge$ chickens, with chickens being comparatively resistant. Other domestic animals, that is, 3- to 12-week-old pigs, pregnant sows, and 1- to 6-month-old calves, also were found to be highly susceptible. For such livestock, the order was pigs, pregnant sows and calves $\wedge$ fattening pigs and mature cattle sheep. Sheep were found to be unusually resistant. The male of the species of rats and ducks has been shown to be more susceptible than the female (Butler, 1964 b; Barnes and Butler, 1964; Wogan, 1966). However, Butler and Wigglesworth (1966) found that female rats in the latter part of pregnancy have greatly increased susceptibility. Most breeds of chicken are much less susceptible than turkey poults, but in South Africa the New Hampshire breed was observed to be much more susceptible than Cornish Game, White Leghorn, or Rhode Island Red (Abrams, 1965).

A wide variety of experimental animals — wild and domestic — have been tested. Marked variations in susceptibility to aflatoxicosis occurred from species to species. Ducklings, trout, rats, rabbits, guinea pigs, dogs, and monkeys are extremely susceptible to minute amounts of aflatoxin. Levels in the order of 1 p.p.m. or less of aflatoxin in their diet have resulted in acute aflatoxicosis. In rainbow trout, approximately one-third of the population developed hepatoma within a year when fed 0.5 p.p.b. aflatoxin. Also, after 20 months on test, trout receiving 2 p.p.b. B_1 had 60 to 70 percent tumor incidence and those receiving 20 p.p.b. had 90 to 100 percent. On the other hand, young silver, or Coho, salmon (Oncorhnychus kisutch) and channel catfish (Hastings, 1969) appear to tolerate single doses of up to 15 mg./kg. body weight of B_1 with minimal liver damage. Hamsters, mice, and sheep also appear to be resistant to levels of more than 20 mg./kg. (Smith, 1969).

With all species, the first clinical signs of disease are weight loss and lack of appetite. Except for general unthriftiness, signs of disease are usually unnoticeable until a few days before death at which time the animal appears listless, develops ataxia, and finally becomes recumbent or moribund.

It has been demonstrated that aflatoxin is a potent hepatic-carcinogen for ducks (Newberne et al., 1964), rats (Newberne, 1965) and rainbow trout (Ashley et al., 1965). According to Ashley (1969), the hepatoma induced by aflatoxin ordinarily is classified as a minimal deviation tumor or liver cell adenoma. In their earliest stages, hepatomas brought about by the ingestion of aflatoxin appear as slightly elevated or subsurface gray to yellowish 1 to 2 mm. nodules in the liver parenchyma while more advanced tumors are generally firm, yellowish, and more or less lobulated nodes. Carcinomas that metastasize are not uncommon.

Molds are commonly found on many foods where they may be present incidentally, or, in more limited instances, intentional ingredients of the final product. A large number of different kinds of molds were found on the surfaces of country cured hams and "fermented" sausages (Ayres et al., 1967; Bullerman and Ayres, 1968; Strzelecki et al., 1969; Sutic et al., 1972). Among those fungi isolated from heavily molded hams or salami were strains of Aspergillus amstelodami, A. chevalieri, A. flavus, A. ochraceus, A. repens, A. ruber, A. tamari, A. wentii, penicillium frequentans, P. puberulm and P. variable all of which, according to studies of others (Hodges et al., 1964; Kulick and Holaday, 1966) contain strains that might be toxinogenic. Since the presence of meat of these molds conceivably constitutes a hazard to human health, isolates were screened for the production of aflatoxin or, in the case of A. ochraceus for ochratoxin.

Of the aspergillus strains tested, only the A. flavus isolates produced chloroform-extractable, fluorescent compounds which coincided with aflatoxin standards on thin layer chromato-plates (TLC). One of these organisms recovered from a commercial sample of Italian-type salami has been deposited in the culture collection of the Northern Regional Research Laboratory, USDA, Peoria, Ill., under accession number NRRL A-16100.

To verify that the fluorescent compounds produced by the A. flavus organisms were definitely aflatoxins, the compounds were purified, and separated by using preparative thin-layer chromatography. When compared with standard aflatoxins, the fluorescent compounds coincided exactly and, in addition, their ultraviolet and infrared absorption spectra were the same as those of the authentic aflatoxins.

Further confirmation was obtained by using bio-assay techniques. The method of Verrett et al. (1964) was employed to determine toxicity to the chick embryo. Inoculation of 0.025 ug. per fertile egg before incubation resulted in death of the embryo. Administration of the extract to 1-day-old ducklings per os or via stomach tube resulted in bile-duct cell proliferation typical of aflatoxin B_1 . Data obtained from 4-day-old chick embryos indicated that the fraction corresponding to aflatoxin B_1 killed the embryos at the highest dilution (0.02, ug. per embryo) within 3 days after administration via the air sac (tests performed by G. Chassis and confirmed by D. E. Tyler).

When pure cultures of Aspergillus flavus A-16100 were inoculated on Italian and Hungarian sausages, minimal amounts of aflatoxin were produced in Italian-type salami stored for 6 weeks at 15° C. and at relative humidities of 65, 75, and 38 percent, but much greater amounts were produced at 20° C. It is interesting that no aflatoxin was produced during the first 4 weeks on any of the salami held at 15° C. but on Italian-type salami stored at 20° C., the amount of aflatoxin increased during the first 3 weeks and decreased thereafter. The cause of this degradation is not known.

CONCLUSION

Although aspergilli are common flora on aged and cured meats, the identification of toxigenic strains of A. flavus by other investigators has not reported from this source. Yet, such strains were detected in aged cured hams and on Italian-type salami. Had these molds been provided with appropriate conditions for production of toxin, they could have produced a real hazard to foods. There is no definite evidence to date that aged or cured meats or both contain harmful amounts of mycotoxin.

OCHRATOXIN

Another toxigenic group within the genus Aspergillus, A. ochraceus, is also widely distributed in nature. In keeping with trivial names used for other mycotoxins, the toxic principle has been termed ochratoxin. Ochratoxins B and C constitute and dichloro and ethyl ester derivatives of the parent compound (ochratoxin A) and seemingly these ester compounds have a toxicity similar to that of ochratoxin A.

A survey reported by Scott (1965) of the microflora of South African legume and cereal products showed 46 strains of 22 species of fungi to be toxic for ducklings. Among these, three of five strains of A. ochraceus Wilhelm proved to be toxigenic. One of the most toxic strains (K-804) was isolated from sorghum grain. Later, Van Walbelk et al., (1968) isolated a Penicillium sp. from packaged ham that produced ochratoxin and an A. ochraceus from Brazil nuts that produced ochratoxin A and small amounts of aflatoxin. The production of aflatoxin by organisms other than A. flavus and A. parasiticus has been questioned by Wilson et al., (1968). Shotwell et al., (1969 a,b) found ochratoxin A to be a natural contaminant in a sample of poor grade corn. Four of five strains of A. ochraceus Wilhelm found in peanuts by Doupnik and Peckham were toxic to day-old cockerels. At least two other members of the A. ochraceus group, A. sulphureus and A. melleus, have been found to elaborate ochratoxin (Lai et al., 1968, 1970).

An investigation of the fate of ochratoxin A when injected into male rats has indicated that the compound it metabolized to isocoumarin and an unidentified green fluorescent metabolite (Nel and Purchase, 1968). All three compounds were present in urine and feces of rats after a single dosage of toxin.

According to Christensen et al., (1962) A. ochraceus Wilhelm invaded grain with a moisture content of more than 16 percent at 20 to 25 percent. In a further study, Christensen et al., (1967) investigated the microflora of red and black peppers. The predominant fungi were found to be A. ochraceus and A. flavus. Autoclaved cornmeal was rendered toxic to both ducklings and rats by the inoculation with A. ochraceus Wilhelm isolated from the pepper.

In the Far East, A. ochraceus Wilhelm and related species were reported as constituents characteristic of the microflora of fermented fish preparations; for example, katsuobushi by Yukawa (1911) and katsuoshuto by Kinosita et al., (1968). In the U.S.A., Patent No. 1,313,209 covers the use of A. ochraceus for its ability to induce a desirable change during the fermentation of coffee.

Several strains of A. ochraceus Wilhelm have been isolated, that is, from stored cottonseed by Christian et al., (1949), from citrus fruit by Doitge (1950), from peanuts by Austwick and Ayerst (1963), from South Africa stock feeds by Van Warmelo (1967), from flue-cured tobacco by Forgacs and Barnes (1968) and from several types of cigarette tobacco by Scheportz et al., (1969).

Strzelecki et al., (1969) working with two strains of A. ochraceus recovered from moldy ham, screened these organisms for ochratoxin production by the method of Eppley (1968). Thin-layered chromatograms of the unknown samples were compared to extracts from a known ochratoxin producer and ochratoxin standard provided by A. D. Campbell, Federal Food and Drug Administration, Washington, D. C. Both strains were found negative for ochratoxin production. Later, however, Escher et al., (1972) found that a strain of A. ochraceus isolated by Sutic et al., (1972) from moldy ham was able to produce ochratoxin A and B on rice and on peanut meal. On rice, 6 ug./g. ochratoxin A and 15 ug./g. ochratoxin B were found; on peanut meal, yields were 1 ug./g. ochratoxin A and 4 ug./g. ochratoxin B. Whether a toxin producing strain of A. ochraceus is also able to produce ochratoxins on ham itself has not been investigated.

Early workers reported that ochratoxin had about the same order of toxicity as did aflatoxin B_1 that is, an LD_{50} of ± 25 ug./duckling (Van der Merwe et al., 1965), but this has been revised to about LD_{50} of ± 250 ug./duckling (Purchase and Nel, 1976).

PENICILLIUM MYCOTOXINS

Citreoviridin. — Several species of the genus Penicillium produce toxic metabolites in moldy or "yellowed rice." One of these, P. toxicarium identified by Miyake in 1938 (Miyake et al., 1970), and since determined to be the same as P. citreoviride Biourge (Saito et al., 1971), causes paralysis as well as respiratory and circulatory disturbances in animals of a type that resembles symptoms of beriberi in man. The toxic moiety, citreoviridin, is a yellow fluorescent polyene that localizes in the central nervous system and causes respiratory paralysis. It is mildly toxic — the minimum lethal dose for rats being 8 to 30 mg./kg. depending upon the route of administration.

P. islandicum metabolites. — P. islandicum Sopp was found by Tsunoda (1951) to cause yellowing or "yellowesis" of domestic and imported rice. The organism produces islanditoxin, a colorless hydrophilic chlorine — containing peptide (cyclochlorotine) $C_{25}H_{36}O_8Cl_2$ and luteoskyrine, a lipophilic 1,1-bis(2,4,5,8-tetrahydroxy)-7-methyl-2,3-dihydranthriquinone. Islanditoxin is extremely hepatotoxic, causing severe liver damage, hemorrhaging and death. Miyake and Saito (1965) reported that LD_{50} values for 10 g. mice range from 4.75 ug. subcataneously to 65.5 ug., orally. Saito (1959) suggested that this toxin interferes with carbohydrate metabolism by causing the disappearance of glycogen granules in the injured liver. By contrast, luteoskyrin is slow acting and larger dosages are necessary to elicit pathological response. The LD_{50} for 10 g. mice is 1.47 mg. subcutaneously and 2.21 mg./10 g. orally (Miyake and Saito, 1965). Liver damage caused by luteoskyrin is of the so-called acinus central cytotoxic pattern characterized by centrilobular necrosis of the liver and diffuse fatty metamorphosis of the liver cells. Luteoskyrin is thought to bind to DNA and cause pigment changes and alter activity of DNA-dependent RNA polymerase (Ueno et al., 1966, 1967 a,b, 1968).

Rubratoxins. — P. rubrum was isolated from corn toxic to pigs, mice, horses, and a male goat by Burnside et al., (1957) and has repeatedly been isolated from moldy feeds. Yet the nature of the toxicity of rubratoxin is not clear. The role that the organism plays in naturally occurring disease is still in doubt (Moss, 1971). There is the possibility that the rubratoxins may potentiate the activity of more dangerous compounds, such as the aflatoxins (Wogan and Mateles, 1968; Moss, 1971). Under laboratory conditions two toxic metabolites are produced — rubratoxins A and B. Moss et al., (1968, 1969) considered rubratoxin B to be the bisanhydride $C_{26}H_{30}11$ and rubratoxin A to have the same structure with one of the anhydride groups reduced to the lactol. Rubratoxin B is the major metabolite and is a higher homolog of byssochlamic acid and probably is produced by the head-to-tail, head-to-tail coupling of two C_{13} units.

Patulin. — The antibiotic patulin is a lactone inetabolite of several species of the genera Penicillium and Aspergillus commonly found on cereal grains. Byssochlomys nevea also has been found to produce patulin. Patulin is toxic to mice, having LD_{50} (oral) of 35mg./kg. in mice. Livestock consuming mold-contaminated feed develop toxicosis. When administered subcutaneously twice weekly, patulin produced malignant tumors in rats at the site of injection. This antibiotic blocks respiration in many bacteria, protozoa, and vertebrate tissues; also, it may alter cellular membrane permeability so as to prevent ion transport.

Penicillic acid. — Penicillic acid is another toxic lactone antibiotic to which carcinogenicity has been ascribed. Since the LD_{50} of this compound in mice is 110 mg./kg., one might question the likelihood of its occurrence in nature at a level that would present a toxic hazard.

There is no question but that the importance of Penicillium mycotoxin in animal feeds is of critical and urgent importance. Yet the significance of Penicillium spp. contamination in human foodstuffs remains to be determined.

Citrinin. — P. citrinum Thom has been found in all the major rice producing countries of the world and frequently has been isolated from polished rice that is yellow in color and fluoresces when viewed under UV light. It is believed that the toxin produced by this fungus, citrinin, is a slow acting nephrotoxin, $C_{13}H_{14}O_5$, having an LD_{50} for mice of 35 mg./kg. when given subcutaneously.

Cyclopiazonic acid. — Albright et al., (1964) isolated a strain of P. cyclopium Westling from peanuts and investigations by Holzapfel (1968) indicated that P. cyclopium caused acute toxicosis in ducklings and rats. The latter worker determined that cyclopiazonic acid was the toxic moiety involved. At about the same time, Wilson et al., (1968) recovered P. cyclopium Westling as a contaminant of feedstuffs involved in two outbreaks of disease among farm animals. Cyclopiazonic acid is a hydrophilic, colorless compound.

T-2 TOXINS

Fusarium tricinctum produces highly toxic scirpine metabolites, tricothecanes or T-2 toxins at low temperatures (Gilgan et al., 1966; Bamburg et al., 1968; Saito and Okubo, 1970). F. tricinctum was found to cause severe dermal irritation when applied to the skin of experimental animals (Tatsuno et al., 1968; Ueno et al., 1970) and to be lethal when injected intramuscularly (Grove et al., 1970). Laboratory workers frequently suffer serious dermal reaction from inadvertant contact with extracts containing these compounds (Grove et al., 1970). Snyder and Hansen (1945) considered F. tricinctum and F. poac, F. chlamydosporium, and F. sporotrichioides synonymous. Types of responses to the trichothecanes suggest that alimentary toxic aleukia (ATA) may be caused by these compounds. ATA may progress in four chemical stages from local irritation of mucous membranes of the cavity and pharynx to a hemorrhagic diathesis with anemia and severe leukopenia upon continued ingestion of toxic food.

The genus Tricothecium includes species which form tricothecanes closely related to those from F. tricinctum and other fusaria. T. roseum produces trichothecin (Saito and Okubo, 1970). T. viride produces trichodermin a closely related compound. Trichothecin was reported to have an LD_{50} of 300 mg./kg. for mice.

PSORALENS

Furocoumarin compounds, more commonly known as psoralens, occurring in the leaves, stems, roots, and fruit of certain plants, have been reported to be responsible for a dermatitis in man. A blistering skin disorder has been reported to be common among celery workers coming into contact with plants infected by the fungus Sclerotinia sclerotorium. An unique characteristic for lesion production is that, following mold contact, the skin must also be exposed to sunlight or to ultraviolet radiation in the range between 320 and 380 mu (Sans, 1941; Pathok et al., 1962). Without such exposure within a few minutes of the time of contamination, lesions are not produced.

Scheel et al., (1963) isolated and characterized two phototoxic furanocoumarins, 8-methoxypsoralen (xanthotoxin) and 4,5',8-trimethylpsoralen, from diseased celery (pink rot) but did not detect these compounds in undiseased plants. Some years earlier Musajo (1954) had isolated a photoxic furanocoumarin from celery (bergapten or 5-methoxypsoralen). Since the mold Sclerotinia sclerotorium can attack almost any kind of horticultural crop, it is one of the most widely distributed pathogens in the vegetable world. Wu et al., (1972) have shown that, under experimental conditions, xanthotoxin and bergapten were produced by the fungus only in the rotted areas and that for production of the two psoralens, the concurrent metabolism of celery and of S. sclerotorium is required. These compounds were not produced at a temperature lower than 15° C. on celery or on artificial media nor were they formed on the non-metabolizing vegetable. Similarly, although the mold grew well on carrots, sweet potatoes, artichokes, oranges, cucumbers, and turnips, no psoralen production could be detected in these commodities.

Temperature, available moisture, oxygen tension, and light intensity influence the growth of fungi in stored foods and feeds. Mechanical damage in harvesting or damage by insects, as well as improper drying and storage, are important factors favoring contamination and toxin production. Contamination of peanuts occurs most often during the drying period after nuts are lifted from the ground (Schroeder and Ashworth, 1965). The optmal temperature and time for aflatoxin production by A. flavus on sterilized peanuts was found by Diener and Davis (1966) to be 25° C. (77° F.) for 7 to 9 days. The organism grows poorly at temperatures lower than 13° C. (55° F.) and little aflatoxin is produced when commodities are stored at low temperature. On the other hand, several Fusarium and Penicillium species grow well at low temperatures and, in fact, the production of their toxic moieties is favored by such treatment.

Similarly moisture content is of critical importance. Tsunoda (1970) observed that when the moisture level in stored rice was between 14 and 15 percent, Aspergillus glaucus and Penicillium citreoviride were able to grow. When the moisture content of rice grain was kept below 14 percent, no mold growth developed for at least a year. Safe storage for groundnuts requires less than 70 percent relative humidity. Sorenson et al., (1967) reported similar time-moisture relations for rice storage.

Growth of molds is retarded by reduced oxygen tension. Most fungi are highly aerobic and require at least 1 percent oxygen for growth.

The relation of different mycotoxins to light is of interest. For example, photodecomposition of aflatoxin B_1 is known to occur at fairly low light intensity (Andrellos et al., 1967). On the other hand, the psoralins seem to be inactive without sunlight; wavelengths of 340 to 380 mu are essential for toxicity.

SALMONELLA–THE CYCLIC PROBLEM

by
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For many years, Public Health authorities have known that food poisoning, in its many forms, is among the most common illnesses in United States. Although most cases are not reported, these authorities believe that the figure may be as high as 10 million cases a year. The most common types of food poisoning are salmonellosis, Clostridium perfringens food poisoning, and staphylococcus food poisoning.

Our purpose today is to discuss the problem of salmonellosis as it relates to foods and to suggest areas of improvement for the future.

Salmonella infection, salmonellosis, is almost always caused by the ingestion of contaminated materials. Gastro-enteritis occurs after an incubation period of from 8 to 48 hours. This short interval suggests that large numbers of the organisms are usually ingested. The onset is nearly always sudden and may be characterized by headache, chills, and often abdominal pain. Nausea, vomiting, and diarrhea follow with a rise in temperature; prostration develops which lasts from 1 to 4 days. Blood cultures are usually negative, but frequently organisms can be isolated from the feces and occasionally from the vomitus.

Over the past 30 years, salmonellosis has become an expanding problem in the United States. Isolation of salmonellae from human sources has been steadily increasing. It has been reported that for every case of salmonellosis, there are from 5 to 20 unreported and diagnosed cases. Human morbidity in the United States due to salmonellae infection is conservatively estimated at 2 million cases per year. Certainly, part of this increasing incidence can be attributed to more sensitive diagnosis, to better isolation media, improved identification techniques, and more efficient reporting.

In the past, salmonellosis has usually resulted from man's own contamination of his environment. As long as the environment was relatively restricted, no serious harm was done. The ailment would run its course through the community; it would usually be mistaken for intestional flu or an upset stomach, and would rarely have serious consequences. If, however, the community has a hospital, a school, or a nursing home for the elderly, an outbreak of salmonellosis can be far more serious. The resulting gastroenteritis can kill the very young as well as the old.

There is no way to measure the cost of salmonellosis to the American economy, but on the basis of a few known examples it can be considered to be at least $300 million. It is responsible for cost in medical care, lost income, livestock and poultry losses, and testing and control programs in the food processing area.

Salmonellosis is an infection caused by bacteria of the genus Salmonella. The genus is composed of motile bacteria that conform to the definitions of the family Enterobacteriaceae and the tribe Salmonelleae. "Salmonella are G-Rods that are either motile by means of peritrichous flagella or nonmotile. Gelatin not liquified, milk petonized, acetylmethylcarbinol not produced, urea not hydrolyzed, nor does it grow in medium containing potassium cyanide. Lactose, sucrose, and salicin are not ordinarily attacked. Found in bodies of warmblooded animals, including man, and occasionally in reptiles, frequently found in the food eaten by these animals."

The above definition is for a typical strain of Salmonella. Occasionally an aberrant strain of one of the commonly occurring Salmonella may be seen. The aberrition may be lactose fermentation or another single test.

The Sub-committee on Enterobacteriaceae of the American Society for Microbiology has proposed that three species of Salmonella recognized are: Salmonella cholerasuis, the type species, Salmonella typhi and Salmonella enteritidis. All salmonellae other than S. cholerasuis and S. typhi are considered to be serotypes of S. enteritidis.

Currently we recognize about 1,300 varieties or serotypes of Salmonella, which are distinguisable from one another by serological means.

These 1,300 serotypes fall into three major groups with respect to their distribution and relationship to human disease. The first group contains those organisms that are primarily human pathogens. Salmonella typhi is an important member of this group. The second group consist of those organisms that are primarily pathogens for animals and birds but may cause disease in man. The majority of salmonellae are found in this group. S. typhimurium, a member of this group, has been the most frequent isolate in the United States in recent years. The salmonellae that are known to be pathogenic only for animals comprise the third grouping. S. gallinarum and S. pullorum are the most important.

The third group is rapidly becoming smaller as more of its members are being found to cause disease in man. The second group is expanding because of the large number of salmonellae that are being isolated from both man and animal. Consequently, it is generally accepted that any Salmonella serotype is potentially hazardous to man.

Of the 1,300 presently known, Salmonella species and serotypes, 33 account for almost 90 percent of the isolates from humans and approximately 80 percent of the isolates from nonhuman sources such as animals, food, and water. Salmonella typhimurium and S. typhimurium var. copenhagen rank first in the 10 most common serotypes isolated from human and nonhuman sources with S. enteritidis second in human isolations and S. anatum that position in nonhuman isolates.

Most salmonellae infections begin in the intestional tract following ingestion of the casual organism. In most recognized outbreaks, the usual vehicle is food. In sporadic cases, however, the vehicle is rarely identified. In these cases, as well as hospital-associated cases, there is convincing evidence of person-to-person or person-to-formite—to person transmission, thus emphasizing the importance of personal hygiene and the human carrier state.

Much has been written and spoken concerning the importance of the human carrier in the transmission of salmonellosis. Most of us are well-informed as to the permanent carrier status of those who excrete S. typhi because of chronic infection of the biliary or urinary tract. It is also clear that permanent carriers of unadapted salmonellae are rare, found mainly in persons in whom Salmonella septicemia occurred. The long-term carrier state (6 months or more) following an infection is commonly realized.

The asymptomatic carriers who have no history of diarrheal disease have been discovered only after routine examination of fecal specimens from food handlers. The number of asymptomatic carriers is unknown because of the short period of time. The bacteria persist and excretion of the salmonellae is intermittant. Certain professional food handlers, including caterers, show a higher carrier rate than persons in other occupations.

The presence of host-adapted salmonellae, such as S. typhi, in market meats from areas of high endemicity emphasizes the role of the human carrier in contamination of food and transmission of salmonellae. This is further emphasized by outbreaks of salmonellosis from smoked fish and watermelon, foods in which salmonellae are not indigenous.

Infants frequently become long-term carriers and may harbor the bacteria for longer periods than adults. They may also transmit infection to other members of the family.

Salmonellae are found in dust. It has been shown that salmonellae occur in the upper respiratory tract before the onset of gastro-intestional symptoms, indicating airborne infection.

Transmission of man-to-man may be direct by means of the fecal to oral route, it may be airborne, it may be indirect by means of foods, toys, towels, contaminated toilet seats, and many other objects. All these avenues may occur in the household, between families, in restaurants, and in institutions such as hospitals. Transmission of unadapted and host-adapted seratypes have occasionally been isolated from animals, such as sheep, horses, dogs, chickens, and hogs.

Water is not a frequent vehicle of salmonellosis because of the efficacy of modern water treatment processes. Untreated water supplies may be contaminated, and surface waters containing human or animal waste show the greatest likelihood of salmonellae contamination.

Pharmaceutical and enzyme preparations from animal organs represent a special problem. The raw materials for granulated pepsin, thyroid powder, pancreatin, and a red coloring, made from a cochineal insect, used for intestinal studies have been shown to contain salmonellae.

Many outbreaks, particularly among children, have been traced to sources such as birds, reptiles, dogs, cats, chicks, ducklings, flies, roaches, ticks, fleas, and other insects.

Dr. Lennington, in his recent report to the FDA/Pet Food Institute Seminar cited several cases of salmonellae transmission to man from dogs. Most heavily involved were children. Dog food, such as dehydrated dog meals, dog candy, and dog biscuits were incriminated as the vector.

Turtles have been incriminated as the vector of several outbreaks of salmonellosis over the past 5 years. The problem has reached such proportions that the State of Washington in 1968 and Dade County, have approved an ordinance prohibiting the sale of salmonellae-infected turtles.

Since 1885 the primary route for salmonellosis is from livestock and poultry to man. It has been suggested that up to 50 percent of raw meat purchased in retail markets is contaminated with salmonellae. Among fresh, red meats the major problem exists with pork and veal. Surface and interior contamination of hogs and cattle constitute the two main areas of the salmonellae problem. Surface contamination of carcasses and abattoir equipment is quite common. One area in which both surface and interior contamination may occur is in the holding pens. Considerable exposure to salmonellae takes place here through contact with feces and contaminated drinking water. Relatively high counts were also detected in the hog feces before slaughter.

The depilitating operation following scalding is another major source of contamination. Subsequent water rinses and the plant atmosphere were also found to contain salmonellae.

The frequency with which salmonellae are isolated from domestic fowl indicates that these animals probably constitute the largest single reservoir of these bacteria. Two-thirds of the cultures isolated from animals in the United States were recovered from domestic fowl. It has been shown that a high incidence of a given type of Salmonella in poultry in a certain locality is accompanied by a high incidence of the same type in man.

Many studies have established the prevalence of salmonellae in poultry on the farm, during processing, and in retail markets. The origin of Salmonella contamination is frequently difficult to determine. It may arise from a bird infected by a congenital or feed source, during processing, or during handling at the retail or consumer level. As with raw eggs, the nature of the product precludes any significant heat treatment or kill step to eliminate the organism.

Young birds surviving salmonellosis on the farm may become carriers and perpetuate the disease cycle. Once the organism has been eliminated from the flocks, adequate sanitation must be taken to prevent recontamination. During processing, plant design and sanitation are necessary to prevent contamination from the colon and during defeathering, eviscerating, chilling, and ice bath operations. Proper storage must be observed through shipping and retail market channels.

Although the Food and Drug Administration (FDA) and the U.S. Department of Agriculture (USDA) require that liquid, frozen, or dried eggs be pasteurized or subjected to an equivalent treatment, the FDA found that about 25 percent of all shipments examined were contaminated with salmonellae. In general, the housewife's use of the raw egg entails a heating step sufficient to kill the organism. However, contamination of other materials arise when these eggs are used in other preparations, or when contamination of other materials arise from contact with infected egg shell surfaces.

Hen's eggs are normally sterile. However, after the egg is laid, contamination may occur with microbes from the soil, from hen's feces, from the nest, and from contaminated detergent washings.

Superior sanitation in the handling of eggs from the farm to the consumer should be stressed to reduce the occurrence of salmonellae in eggs. The contamination of feeds is another area in which adequate surveillance is needed to prevent the infection of hens.

Salmonellae invasion of nonfat dry milk solids, powdered milk, noodles, cake mixes, dried yeast, and yeast tablets are some of the many other food items incriminated in recent salmonellosis outbreaks in the United States.

FDA has concluded that action should be taken to eliminate animal-based sources of infection. The major responsibility for animal infection lies in contaminated feeds and feed-mixes which utilize animal byproducts and thus start the vicious chain. Investigation has shown that such feed additives as processed fishmeal, poultrymeal, meat scraps, and meal are primary sources of salmonellae.

These products are processed without any regard for sanitation, simply because they are intended for animal consumption, and sanitation is considered unnecessary for such purposes.

The processing methods used for these materials intended as feed mixes do not always kill the salmonellae; and even if it did, the product is immediately recontaminated in salmonellae-loaded surroundings.

Animal feed manufacturers insist that they cannot hope to produce salmonellae-free products as long as the ingredients they use arrive salmonellae-laden at their plants.

If voluntary compliance by the suppliers of animal byproducts to recent FDA guidelines and regulatory actions by the FDA result in the delivery of salmonellae-free animal byproducts to feed manufacturers, then we may have reached the stage where the primary chain of contamination of animal-derived food intended for human consumption will be broken.

It would be a mistake to assume that human salmonellosis can be eliminated merely by assuring the absence of the organism from animal-derived foods as they are sold to the consumer. To do this, salmonellosis in domestic animals and contamination of foods during processing must be controlled, and salmonellae in raw products must be destroyed and their growth prevented by processing. Regulatory, surveillance, and educational procedures must be developed for and applied to the task.

The conscientious manufacturer or processor takes all the precautions outlined above to deliver to the consumer a clean, wholesome product, but the housewife in her kitchen may contaminate the product or prepare the product improperly and thereby create new problems.

The ingredients she adds to prepared mixes may contain salmonellae, which exist on eggs, milk, meat, and poultry. If the product is heated sufficiently, no problems will arise. However, many modern foods do not require heating.

Another source of spoilage may be improper preparation. Recipe directions may call for boiling a product, but the housewife may heat only to a slight simmer at the edge of the pot without stirring. Thus, conditions could exist where a product will not reach sufficient temperature to pasteurize it.

Most women take what they feel are reasonable precautions when preparing a meal. Nevertheless, experts say that the majority of food-poisoning cases result from improper food handling in the home. The three most common types of food poisoning can be prevented by proper food handling.

Stress personal hygiene to all members of your family. Anyone with an infectious disease or a skin infection should not be allowed to prepare or serve food. Always wash thoroughly before working with food, after going to the toilet or blowing your nose, and after touching raw meat if you then are to handle other foods. Use utensils rather than hands if possible.

Serve foods soon after they are cooked. If this is impossible, refrigerate them. Decrease the time necessary for cooling large amounts of food by placing them in shallow containers before refrigerating. Keep foods in the refrigerator until they are to be reheated or served. Keep foods hot (temperatures above 140° F.) or cold (temperatures below 40° F.). Foods left at temperatures of from 60° to 120° for 3 or 4 hours may not be safe because bacteria grow rapidly at these temperatures.

Carefully clean all dishes, utensils, and work surfaces with soap and water after each use. Work surfaces can be cleansed of bacteria by using laundry bleach in amounts recommended on the package. Do not use the same surfaces on which you prepared meats or other foods before cooked as work areas on which to further prepare them after cooking unless the surfaces have been thoroughly cleaned. The cooked foods can be recontaminated by the bacteria remaining on the surface.

There can be no doubt that the producer, processor, regulator, and the consumer have made progress. Further progress can be achieved as long as we maintain vigilance and determination to deal with the Salmonella problem.

In closing, may I remind you that one of the favorite dishes in Louisiana and the other Gulf Coast States are the result of seining for shrimp in the Gulf of Mexico. In over 10 years of inspecting the raw green headless shrimp from boats or dockside, I have never found a positive sample for presence of Salmonella. For the past 5 years, samples also have been run on breaded shrimp products from shrimp processors on the Gulf Coast from Miami to Brownsville, Tex. Again I have never found a sample which was positive for Salmonella. Of course, FDA regulations prohibit the release of any of these products if there is the presence of just one salmonella. The built-in safeguard in these products of boiling or frying, or both leads to the destruction of salmonellae, but further contamination by handling is always the problem. It is the shrimp salad, which one usually finds as the culprit, caused by poor sanitation and handling as well as lack of refrigeration for lengthy periods of exposure during warm periods when rapid growth of the organism can occur.

Another Louisiana delicacy, crawfish, is relatively free of salmonellae. In our samplings, we have found only about a 3 percent incidence, due to crawfish obtained from a polluted area which was traced and verified as coming from garbage and outdoor privy run-off.

So, please enjoy our fresh Louisiana shrimp, oysters, and crawfish during your stay with us here in New Orleans, and know that Public

Health records are clean as far as the freshly prepared product is concerned.

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NITRITES AND NITROSAMINES IN RELATION TO FOOD SAFETY

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In view of recent reports of certain nitrosamines in meat and fish treated with sodium or potassium nitrite, it is appropriate at this conference to examine the relationship of nitrosamines and their precursors to the safety of our food supply.

Nitrosamines have been used as intermediates in the drug industry and in the manufacture of other types of compounds. In 1954 Barnes and Magee (1) established the correlation between N-nitrosodimethylamine (DMNA) and liver toxicity. This was extended to the relationship of DMNA to tumor development in a number of test animal species. To date, however, no known incidences of cancer in humans can be related to exposure to nitrosamines, but, since all animal species tested have been sensitive to one or more nitrosamines, it can be anticipated, by extrapolation, that humans, too, are sensitive.

The connection between nitrosamines and the food supply was suggested in 1963-65 when liver toxicity in sheep and mink was found to be produced by N-nitrosodimethylamine present in the fishmeal fed to these animals. It was shown that the DMNA was produced in fishmeal from one or two particular processing plants that added large quantities of nitrite to the fish as a preservative agent, and then dried the fish at high temperatures.

Nitrite is used in the processing of some fish and meat products. More than 2 billion pounds of meat are treated yearly in the form of ham, bacon, frankfurters, and other sausage products. The action of nitrite on meat is threefold: (1) to develop characteristic flavor; (2) to develop characteristic color; and (3) to exert a preservative action against Clostridium botulinum, particularly in semicooked canned products. The first two activities are not related to food safety; therefore, they will be discussed only briefly. The characteristic cured flavor of pork products requires the presence of nitrite. In its absence, only a cooked salt pork flavor is developed. Our studies have shown that there is some interaction between nitrite and smoke in developing the flavor of smoked, cured products; but the nitrite is still necessary for full flavor. Only a few investigations have been carried out on the effect of nitrite on flavor.

There is more concern about nitrite and cured meat pigments, the so-called cosmetic effect. The role of nitrite in developing color has been investigated. Although the fate of nitrite after addition to meat is not fully known, it appears that nitrous acid, formed from nitrite, is reduced to nitric oxide which complexes with metmyoglobin. The nitrosylmetmyoglobin is reduced to nitrosylmyoglobin which forms the red pigment nitrosylhemochrome upon heating.

Canned, cured meat products have enjoyed a long period of safe consumption principally as a result of the inhibitory action of the cure salts against spoilage bacteria, particularly Clostridium botulinum. This organism produces a highly toxic product under the anaerobic conditions of vacuum packaging or canning. While heat will destroy the organisms, the amount necessary to accomplish this would alter the characteristics of the product. Although the growth of clostridia in the product causes unpleasant spoilage, toxin formation is not necessarily correlated with acceptability. Pivnick and others (8) demonstrated that increasing concentrations of nitrite in vacuum-packed hams delay the onset of both toxinogenesis and spoilage. Toxin was present in all samples, except at the highest level of NO_2^-, before 90 percent of the panel judged the hams unacceptable.

Inhibition of the growth of Cl. botulinum in meat products is a complex phenomenon involving interaction of the number of spores present, the concentration of salt in the cure, the concentration of nitrite, and the amount of heat applied. The interdependence of these factors is so great that minor changes in the conditions of one of these parameters may require balancing modifications in all of the others.

A number of outbreaks of botulism from eating improperly processed fish or fish products have occurred in recent years. Control of Cl. botulinum in fresh fish fillets or lightly smoked marine fish, such as salmon, tuna, halibut, and cod, as well as fresh water fish (chub) can be accomplished by the use of nitrite in quantities prescribed by law; that is, a residual of not more than 200 mg./kg. The nitrities, however, if used improperly can be toxic — as is almost any other compound that is ingested. The toxicological level of nitrite is not of immediate concern, but there are at least two potential hazards in the use of this compound. Methemoglobinemia is a known condition and fatal incidents have been reported; however, the formation of nitroso compounds has been described only recently and the degree of danger is still unevaluated.

N-Nitroso compounds are formed by the reaction of nitrites with amines. Classically, only secondary amines reacted to form N-nitrosamines. Amides may also react to form N-nitroso compounds. Tertiary amines were thought not to react to form N-nitrosamines but it has been shown recently that this is true only under very strong acid conditions; at pH 2 to 4 nitrosamines are formed. In model systems nitrosamine formation is optimal at pH 3.5. The rate is dependent on the basicity of the amine, the greater the pK_b, the slower is nitrosation. The rate of nitrosation is proportional to the square of the nitrite concentration and may be increased by various ions, such as SCN^-, Br^-, and I^-. Temperature also affects nitrosation; the rate increasing with increasing temperature.

Nitroso compounds in the food supply are of particular concern because most of them are carcinogenic, and possibly mutagenic and teratogenic as well. They produce tumors of the liver, esophagus, respiratory tract, etc. Many are organ-specific. The toxic or lethal doses have not yet been determined, and this is one of the difficulties in evaluating the problem. Long-term rat feeding studies with pure DMNA in concentrations of 1 p.p.m. led to tumor formation, as did a single dose of 5 p.p.m. This is an area of great concern, requiring considerable investigation.

Another problem in the study of nitrosamines is the analytical aspect. Nitrosamine levels in foods reported to date are in the ug./kg., or p.p.b. range. Extraction of such trace quantities of material from the sample with good recovery is difficult. Contaminating material is often extracted, interfering with quantitation or confirmation. The latter is a critical factor in evaluating reports of nitrosamines in foods. Quantitation methods in common use are Thin Layer Chromatography (TLC) separation with identification by spraying with Griess or Preussmann reagents, or both; polarography; reduction to hydrazines and coupling to give colored derivatives; oxidation to nitramines; and quantitation by Gas Chromatograph (GC), using an alkali flame ionization detector. The only method that can give confirmed nitrosamine identification, at this time, is one that includes mass spectrometry, preferably the GC-MS combination.

The interest in nitroso compounds in foods involves three areas: naturally occurring nitroso compounds, production through processing, and formation in vivo.

Reports of nitrosamines in raw foods are not common. N-Nitroso-4-methylaminobenzaldehyde was identified in a mushroom, Clitocybe suaveolens, but this was grown in a liquid culture medium containing nitrate and malt extract which may have contained amines. The compound apparently was found only in the medium. Ender and Ceh (2) also found traces of nitrosamines in single samples of mushrooms. There is a report of N-nitrosodimethylamine in the fruit of a bush, Solanum incanum growing in the Transkei area of South Africa, particularly where the soil was deficient in molybedenum. A correlation between these factors and the incidence of esophageal cancer in the Bantu people was claimed. This report has not been substantiated in later samples from this area.

N-Nitrosamine has also been reported in raw sable fish and in raw meat in very low concentrations — on the order of 5 ug./kg. The origin of these nitrosamines is not known. Hedler and Marquardt (7) described N-nitrosodiethylamine in various parts of the wheat plant; the grain contained the largest amount. However, the methodology used in these tests was crude and the interpretation of the results is open to question.

Of greatest concern at this time is the possibility of forming nitroso compounds as a result of processes that include the use of nitrite. As indicated previously, N-nitrosodimethylamine was identified in fish meal treated in a manner not consistent with accepted practice.

It was also shown that nitrite reacts with the amines in fish, the rate of reaction being greatest for dimethylamine, then trimethylamine, and slowest for trimethylamine oxide. The trimethylamine oxide is present in comparatively large amounts in marine fish. Trimethylamine and dimethylamine are degradation products. Fresh water fish have considerably less amines and are probably less liable to nitrosamine formation on processing, although N-nitrosodimethylamine has been identified in chub. Fazio and others (3) found DMNA in smoked and nitrate-treated salmon and shad as well as raw and treated sable. Some evidence of nitrosamine formation in smoked kippers and haddock was reported by Ender and Ceh (2). Sen and others (10) found low levels of DMNA, N-nitrosodiethylamine and N-nitrosodipropylamine in some samples of smoked cod, herring, halibut, mackeral, hake, and salmon, but not in any sample not treated with nitrite.

Cured meats, naturally, are prime suspects in formation of nitrosamines. In the earlier literature 400 ug./kg. N-nitrosodiethylamine had been reported in Kasseler meat — a cured product. This is probably the only positive report of substantial quantities of nitrosamines in meat until recently. Ender and Ceh (2) showed some values in meat, but they were less than 5 p.p.m. and were not confirmed. Fazio and others (4) investigated 51 meat samples of various classes and in only one, a ham, was as much as 5 ug./kg. DMNA found and confirmed by Mass Spectrometry (MS). Canned pork luncheon meat, Danish back bacon, English bacon, ham, fresh pork, and fresh beef were analyzed in another study using GC-MS, and no DMNA was found at the 25 ug./kg. level, which was the sensitivity of the procedure. Fiddler and others (5) studied 10 hams — imported, domestic, canned, and fresh. All had levels of apparent DMNA below the sensitivity level of the test. In September 1971 we initiated a survey of nitrosamines in frankfurters and found DMNA in products of two out of eight producers examined. An intensive survey of the products of the two companies showed two samples of frankfurters containing DMNA out of 22 samples of one company and only the initial contaminated sample out of 12 of the second company. Since then C&MS has reported DMNA in three samples of meat products and the FDA identified N-nitrosopyrrolidine, at 40 to 100 ug./kg. levels, in bacon prepared as in the home.

A major problem in isolating and identifying nitrosamines at this time seems to be the erratic manner in which they are produced — they are present in a few samples, not in others. This appears to indicate localized areas in the product where conditions are optimum for nitrosamine formation — high nitrite or amine concentration, pH, temperature, and so forth. We are carrying out studies on the processing of frankfurters in an effort to determine the existence of such conditions.

The question of nitrosamines in some cheese products has been raised. A few European countries permit the addition of nitrate to cheese-milk to prevent undesirable bacterial growth. Microbial reduction of the nitrate results in a supply of nitrite. Several investigators have reported negative results. Only a few nonconfirmed positive reports have been published.

One to three p.p.m. DMNA were found by polarography in samples of a fermented drink used by the native population in a section of Africa where esophageal cancer exists. However, British-produced spirits were also tested and no nitrosamines were found by GC-MS examination, although positive results were indicated by polarography.

The formation of nitrosamines in the gastrointestinal tract following ingestion of nitrites and foods containing amines or amine precursors is another problem that is being investigated. Nitrosated amines have been recovered from the stomachs of test animals and, in other experiments, tumors have been found after feeding certain amines such as morpholine, methylbenzylamine or diethylurea, and nitrite. There are many questions about the validity of the animal experiments, that is, species of animal used, concentrations of test substances, how administered, interpretation of results, etc. Finally, and most important, there is the question of the significance of the amines tested to foods. Proline, hydroxyproline, arginine, and several other amino acids suggested as possible precursors as a result of degradation of proteins, have proved noncarcinogenic in feeding tests at this point. It is possible, however, to nitrosate naturally occurring amines. Proline may be decarboxylated to pyrrolidine, which is nitrosated with little difficulty in model systems. Fiddler and others (6) reported the nitrosation of several naturally occurring quaternary amines under conditions simulating those of frankfurter processing. In model systems neurine, choline, acetylcholine, carnitine, and betaine formed trace amounts of the appropriate N-nitrosamine. The significance of these findings to the human situation with respect to meat is still unknown.

Only one human experiment on gastric nitrosamine formation has been reported. Sander and Sief (9) fed a number of patients diphenylamine and nitrite and identified N-nitrosodiphenylamine in the stomach contents. These patients, however, suffered from conditions in which the pH of the stomach was more alkaline than is normally found, and the amine used (because it forms one of the few noncarcinogenic nitrosamines) happens to nitrosate more readily than the dangerous compounds.

The nitrosamine problem in foods is still a potential problem since no undesirable human cancer incidents have been reported. Involvement of nitrite itself in other conditions, however, is a matter of record. When large quantities of nitrite are ingested, the salt is absorbed from the stomach and some of the nitrite may oxidize hemoglobin in the bloodstream to methemoglobin. Hemoglobin in this form cannot transport oxygen so cyanosis occurs with potentially fatal results. Several incidents of metheglobinemia have been reported in children after eating frankfurters. In fact, in 1955, 10 children in New Orleans developed this condition from locally produced frankfurters and bologna. These products contained extremely high concentrations of nitrite — on the order of 5,000 p.p.m. A classic story of methemoglobenemia in adults is that of "The Eleven Blue Men" in which an outbreak of this condition in 11 men was traced to a restaurant all had frequented. All the men had oatmeal for breakfast, and all added sugar to their oatmeal. It was found that a restaurant employee had inadvertently filled the sugar bowls with sodium nitrite kept by the restaurant for corning beef. Fortunately, however, incidents of methemoglobinemia in children and adults are rare. This condition is of concern primarily in infants under 4 months of age, who are more susceptible because they still have a fetal form of hemoglobin which is apparently more sensitive to nitrite oxidation and because the bacterial population of their gastric tract may reduce nitrates to nitrites more readily than the adult flora.

Nitrites enter the food supply either added, as in cured products, or as a result of reduction of naturally occurring nitrates. This latter is the major source of nitrite. Plants accumulate nitrate as a normal step in their metabolism. The quantity depends on the species, type of fertilizer, light conditions, moisture conditions, trace components, and so forth. Normally, nitrates are reduced to nitrites which are further metabolized as part of the protein cycle of the plant. Many of the green, leafy plants, vegetables, and forage grasses have high concentrations of nitrate. Under proper conditions bacterial reduction of the nitrates to nitrites and nitric oxide gases can occur. Spinach has been implicated in infant methemoglobenemia. This plant is normally rich in nitrate. A number of reports indicate that spinach, either home cooked or commercially prepared, may contain high concentrations of nitrite if left open at room temperature for a number of hours.

Many outbreaks of infant methemoglobinemia have been traced to the water used for drinking and for food preparation. High concentrations of nitrate have been reported in the water supplies in many areas of the United States and Europe. This is particularly true of well-water in rural areas, where nitrate can originate as run-off from fertilized fields or manure piles. Bacterial reduction of nitrate to nitrite may occur in infants with stomach conditions such that bacteria from the lower bowel can grow in the stomach.

Thus, it is evident that, as a result of potential hazards from nitrite in the food supply, further research is required to determine the conditions under which the hazards might exist; and, if they do, how to avoid them. Nitrite plays an important role in the meat and fish industry. Any decisions made with respect to this ingredient must be based on scientific fact and should not be influenced by emotionalism, or we may introduce another set of problems that may be even more difficult to solve.

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NITRATE-NITROGEN IN SOIL AND POTENTIAL EFFECTS ON THE ENVIRONMENT

by
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Introduction

Most of us want to drink pure water. Water, however, is the most universal solvent known and pure water does not exist. When water travels through the soil, it tends to lose the ions that are tightly held by soils and to gain ions that are not held tightly by soils. The nitrate ion belongs to the latter class so that soil water usually is fairly high in nitrate.

Water which is high in nitrate can be harmful in three ways. First, the nitrate can encourage aquatic growth, although nitrate nitrogen seldom is the most limiting factor in such growth. Second, it can cause death of infants under certain conditions, but apparently only when digestive upset occurs. Third, it can contribute to death to livestock from nitrate poisoning. However, it is fair to say that no one has any very accurate idea of how harmful nitrate really is. In Dell City, Tex., for example, natural nitrate levels in water are extremely high and no particular human or livestock problems have been blamed on this condition. Another situation occurs in California where the state health department has raised the standard of nitrate nitrogen from 10 to 20 p.p.m. for the reason that most water is above 10 p.p.m. and has had no ill effects on people.

A number of factors affect the nitrate concentration of water that finally flows into ground or surface water. Among these are the pattern of water movement in the soil, the mineralization of organic nitrogen by soil organisms, the oxygen content of the soil, plant growth and, of course, fertilization.

This paper discusses these factors and some practical steps that can be taken to reduce the level of nitrate in ground and surface waters.

I want to acknowledge the research of several of my colleagues in this paper. They are Seri Sookaskitch, Matthew McMahon, Susan Rhodemyre, and Harold Miller. Without their help, the paper would not have been possible.

Reactions in Nitrate in Soils

In most soils nitrate is not absorbed by clay surfaces so that it is either dissolved in the soil solution when the soil is wet or precipitated as a salt (usually calcium nitrate) when the soil is dry. When rain occurs on a soil, the nitrate in the soil solution is mixed with the rain water and gradually moved down through the soil. A heavy rain on a wet soil moves nitrate much deeper than it does on a dry soil because the soil aggregates in a wet soil are fairly waterproof to rain and most of the nitrate (and water) moves through only a part of the soil volume. In a dry soil, on the other hand, the aggregates continually remove the incoming water from the pores, much as a dry sponge does, so that the depth of water penetration is much less. The mechanism for this is shown in figure 1.

When water moves upward in soils, nitrate also moves in that direction. During most of the growing season in the Southeast, more water is lost by evapo-transpiration than falls in the form of rain. Therefore, there tends to be upward movement of nitrate towards the soil surface. All other soluble salts behave in the same way. Some results for a Maury silt loam at Lexington, Ky., during the 1971 season are shown in figure 2. These results are typical for upland soils with good internal drainage in much of the humid part of the United States.

Anything that reduces evaporation from soil will increase the downward movement of nitrate in soils. Examples are the use of mulches and the formation of natural mulches on clay soils which crack upon drying. Therefore, in both mulched soils and "self-mulching" soils water is conserved, but more nitrate is lost.

The above statement implies that when evaporation becomes low, water will flow through soils rather continuously. This is exactly the case. As soon as cooler weather reduces evaporation, water flow through soils becomes much more important. This is illustrated in

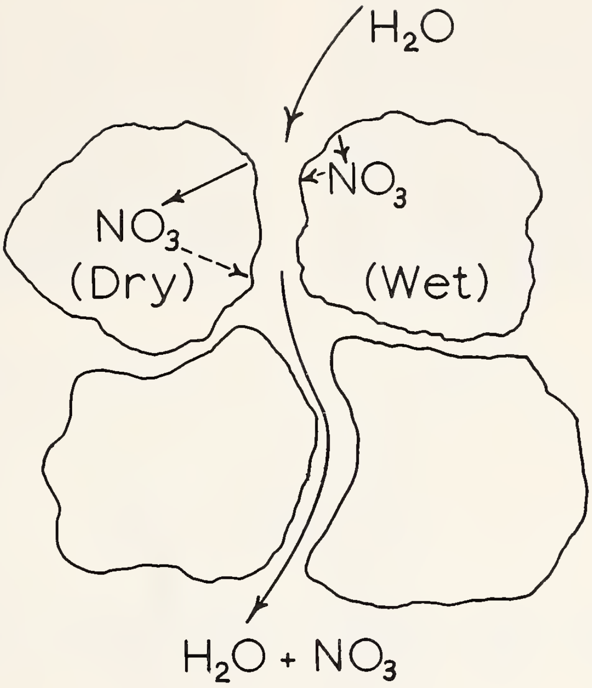

Figure 1. Mechanism of nitrate movement in wet and dry soils

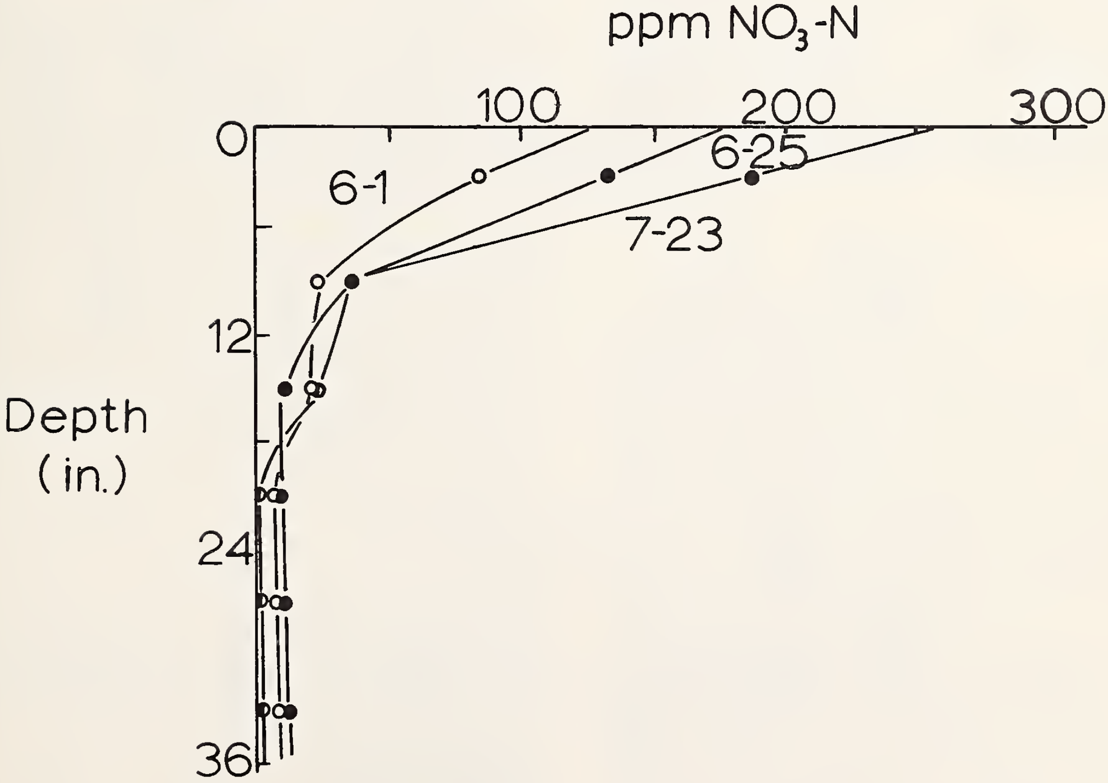

Figure 2. Accumulation of nitrate at the soil surface at Lexington, Ky. summer 1971

figure 3, which shows the rainfall and runoff from a small watershed near Lexington. All the soils in this water shed are extremely permeable so that nearly all the stream flow is a result of underground flow. Notice that the runoff nearly parallels rainfall for the months of November through May but that from June through October, rainfall causes virtually no streamflow. Since nitrate will not move unless water does, this means that there is no leaching in the watershed during June through October. In a rough way, this graph applies to nearly all of the South.

There is not too much variation in the pattern from year to year. Runoff records for the past 10 years on this watershed were compared with rainfall; the results shown in figure 4. Although rainfall varied from 36 to 49 inches, the difference between runoff and rainfall was fairly constant at about 27 inches per year. This suggests that if one knows the rainfall for a given year, he can substract the average evapo-transpiration (27 inches for Central Kentucky) and predict the amount of water available for leaching nitrate.

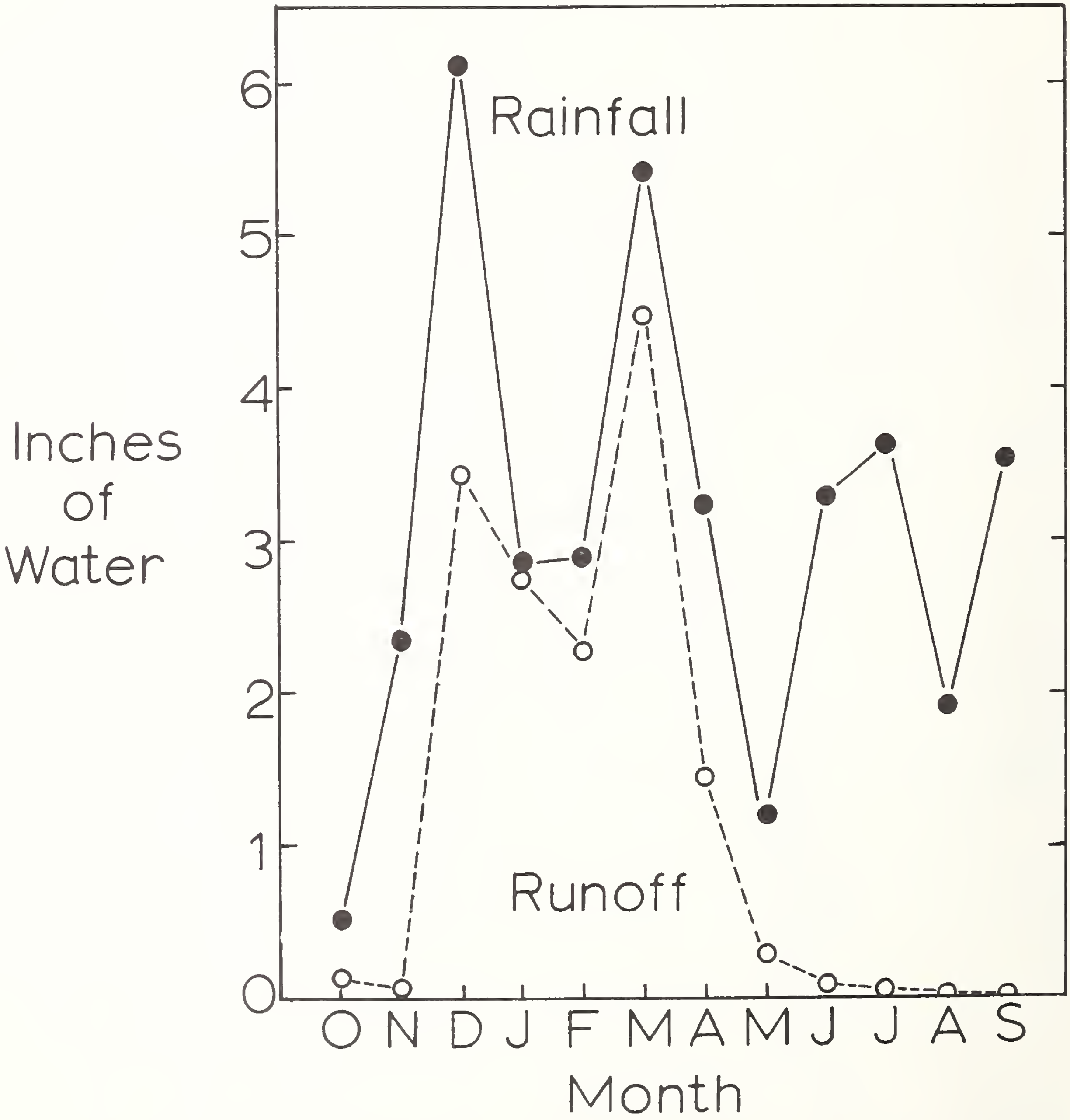

Figure 3. The relation between monthly rainfall and stream runoff for 1964-65, Cave Creek, Lexington, Ky.

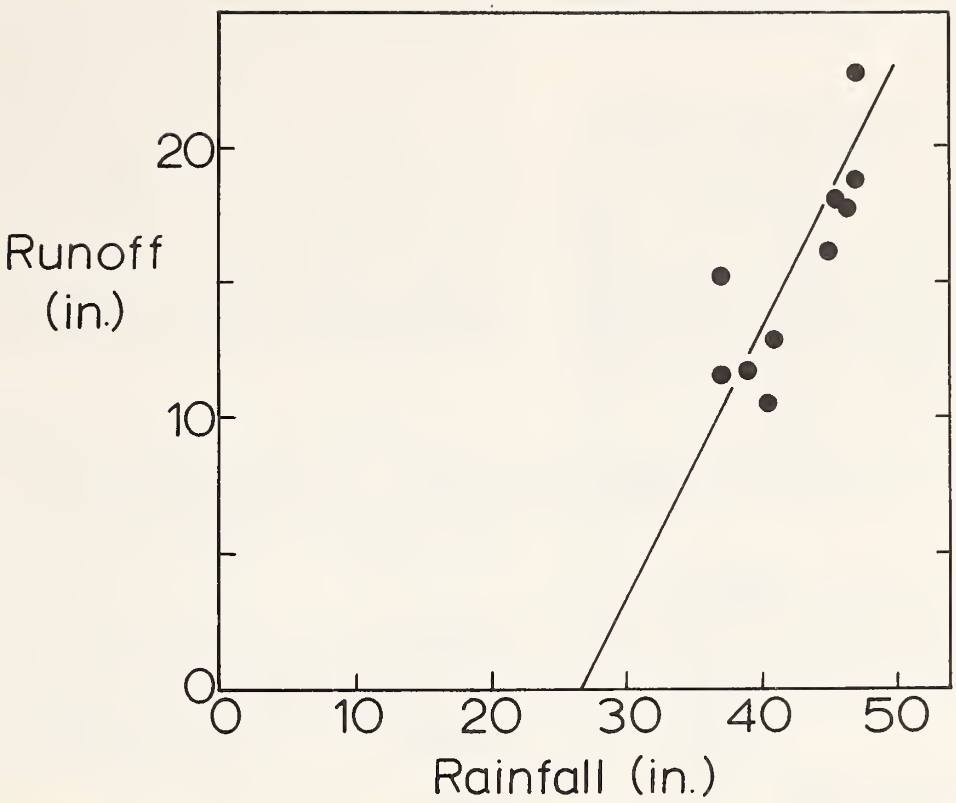

Figure 4. A 10 year relationship between yearly rainfall and runoff, Cave Creek, Lexington, Ky.

If the average yearly value of streamflow is 18 inches and if each foot of soil holds about 4 inches of water, then one can expect about 4½ feet of soil to be leached thoroughly during the cool part of the year. Usually 4½ feet will include the root zone. Therefore, in much of the South, the winter rains will pretty effectively remove the nitrate that is present in the soil after the crop is grown.

There is one specific exception to this, and that is the bright red soils of the southern Piedmont, Limestone valleys and the Coastal Plain. The red color in these soils is due to a high iron oxide content, and iron oxide is positively charged. Because of its positive charge, it holds onto nitrate, but holds onto it fairly weakly. The result is that nitrate, on the average, moves only about half as fast as the water, and does not move entirely out of the root zone during a single winter. A comparison of two soils in Kentucky, one of which was a red soil, showed that the proportion of nitrate nitrogen applied in November that still remained in May was 100 percent for the red soil and 40 percent on a light-colored soil. Similar results have been reported by Boswell and Anderson in Georgia.

To summarize briefly, the amount of nitrate lost is pretty well related to the water available to move through the soil. This can be approximated by stream runoff. Any soil condition which greatly reduces water loss by evaporation will increase leaching. Most leaching occurs in the winter and spring. In most of the South, there is enough water to remove nitrate from the root zone of soils. One exception to this is the red soils, where nitrate movement is slowed. The nitrate carryover in the winter in these soils is appreciable.

Denitrification

In contrast to leaching of nitrate, very little is known about biological denitrification. Apparently, facultative anaerobic bacteria use oxygen gas until it is severely depleted and then turn to other sources of oxygen. Since nitrate is rather easily reduced, it is one of the first to be used. If all the oxygen from nitrate is used, the end product of nitrate metabolism by facultative anaerobes is nitrogen gas. Some scientists believe that all the nitrogen gas in the atmosphere got there from this process so that when fertilizers are made from nitrogen gas we are merely recycling a waste product.

Work here in Louisiana by Patrick showed that the average oxidation potential where nitrate began to be reduced is about +340 millivolts. Most soils have pockets within them that are more reduced than this. It is safe to say that all soils suffer some denitrification every year. Our wetter soils fall below this potential frequently during the growing season and denitrification accounts for a considerable loss of nitrate in these soils.

Besides excessive water and the lack of oxygen that accompanies it, another indispensable requirement for rapid denitrification is rapid bacterial growth. This is accomplished by a high temperature and the presence of easily oxidized organic matter. A good source of this microbial "food" is crop residues that are turned under during soil preparation. The incorporation of residues has long been associated with nitrogen deficiencies in the soil, presumably because of the "immobilization" of mineralized nitrogen in microbial cells during the period of microbial population buildup. There is some evidence to suggest, however, that in wet soils, the nitrogen from residues and the nitrate in the soil are both lost as a result of denitrification. Some results of ours, using ground corn stalks are shown in figure 5. Note that nitrate disappears rapidly and that Kjeldahl (organic) nitrogen also decreases. Soils that tend to be wet and in which crop residues are plentiful are prime locations for denitrification. Since denitrification produces an innocuous waste product, (nitrogen gas) it can be beneficial under some conditions.

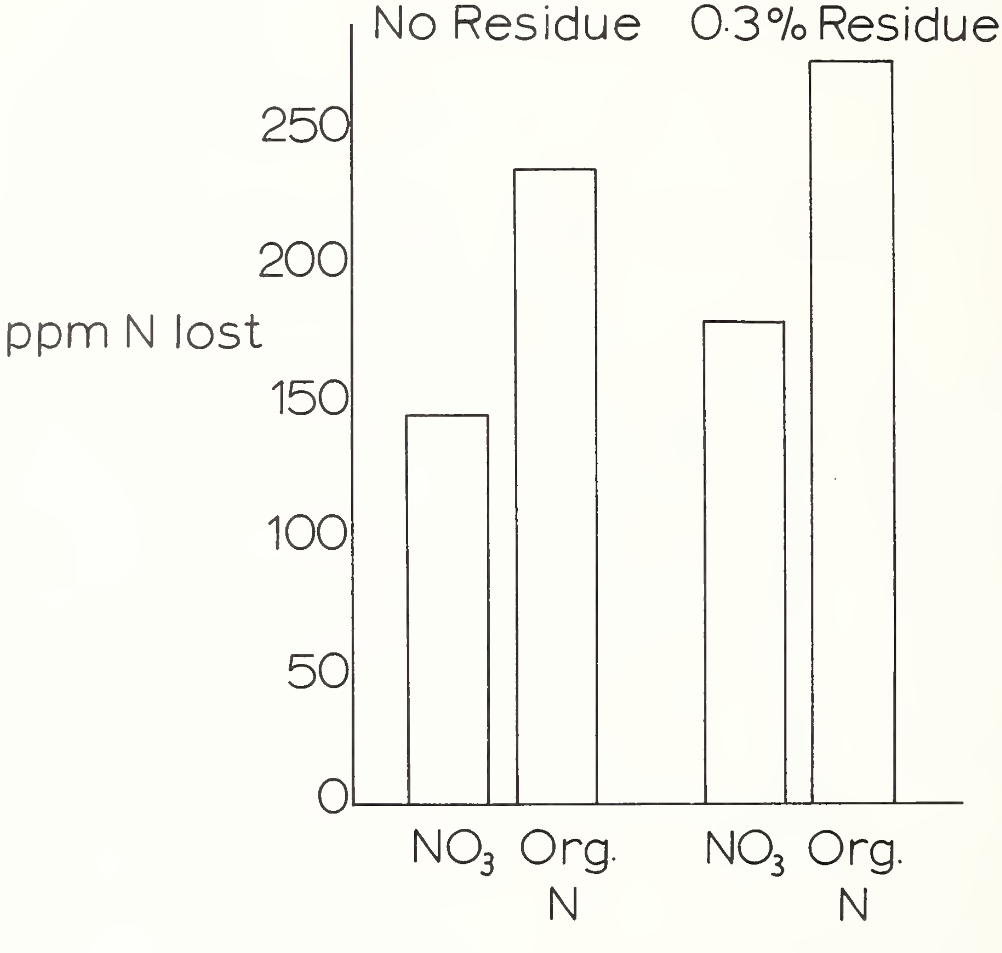

Figure 5. Loss of nitrate and organic nitrogen from a submerged soil, with and without cornstalk residue

Plant Uptake

The removal of nitrogen from soils by plants is roughly proportional to the amount of dry matter produced. Thus, in the case of corn, there is practically no nitrogen removed for a month after planting because the corn is small. However, in the second month after planting, corn grows geometrically and essentially all the nitrogen that the plant needs is removed. Potatoes, small grains and tobacco act much the same. Ideally, we could produce good corn and completely prevent any contamination of water if we added nitrogen fertilizer exactly at the time it is needed. We have some experiments that attempt to do this, but in a practical sense we never will be that exact. Table 1 shows how quickly corn depletes nitrogen in a soil down to the same level as that found in the check. Corn

Table 1. Nitrogen found at two dates in a Melvin soil planted to corn

	NO_3–N lb. per acre	
N RATE (lbs./acre)	MAY 26	JULY 20
0	80	22
100	242	22
200	316	26

only requires a certain level of nitrogen for optimum growth, however, and excess nitrogen above this level will not be taken up. In a fertilized soil with high mineralization and a short corn crop, the results shown in figure 6 are likely to be found. Leaching of this nitrate is a strong probability.

In grass, the situation is quite different. Dry periods stop grass from growing but do not kill it. Then, as rains occur, grass grows again and takes up more nitrogen. Some results for a mixed orchardgrass-bluegrass sod at Lexington are shown in figure 7. Efficiency of added nitrogen in producing protein was close to 100 percent at the highest rate. Under such conditions, very little contamination of water seems likely.

In general, crop removal of nitrogen greatly reduces the nitrate content of the soil. This reduces the likelihood of water contamination and, of course, increases crop growth, which is the objective of nitrogen fertilization in the first place.

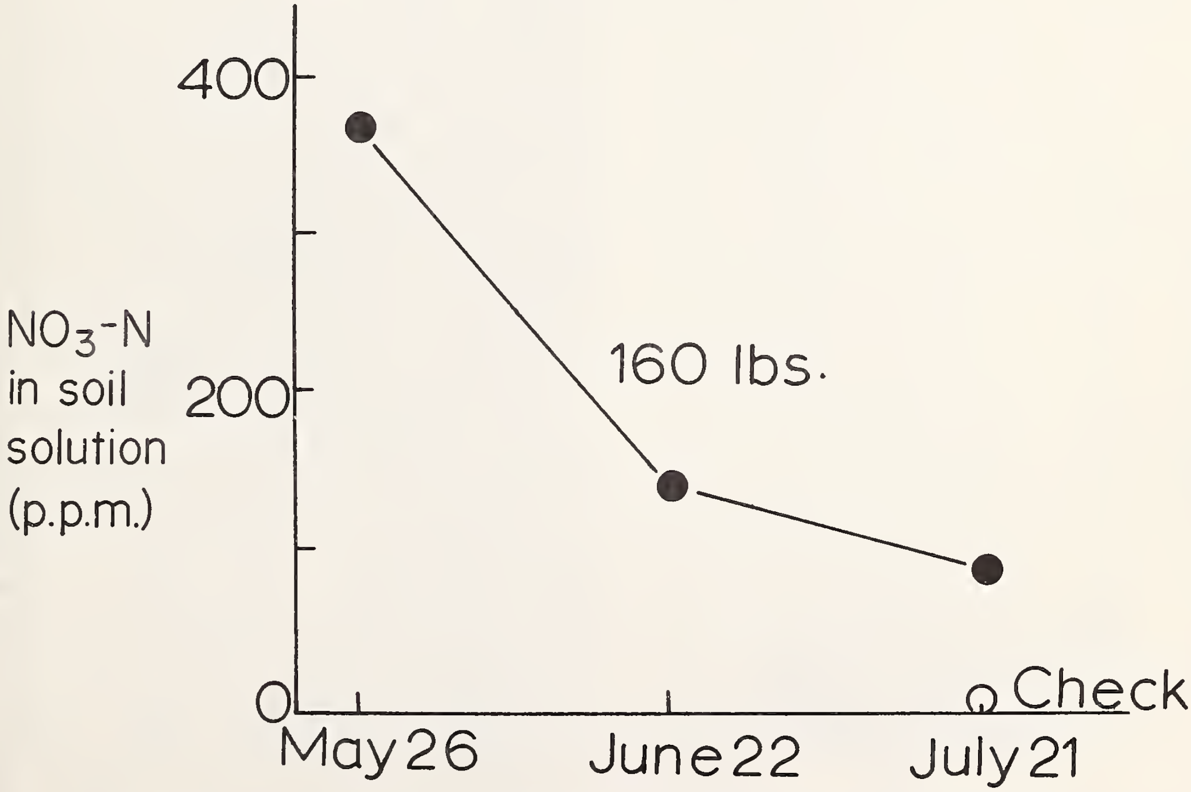

Figure 6. Nitrate-nitrogen in the soil solution in a fertilized Pembroke soil planted to corn, 1970 season, Franklin, Ky.

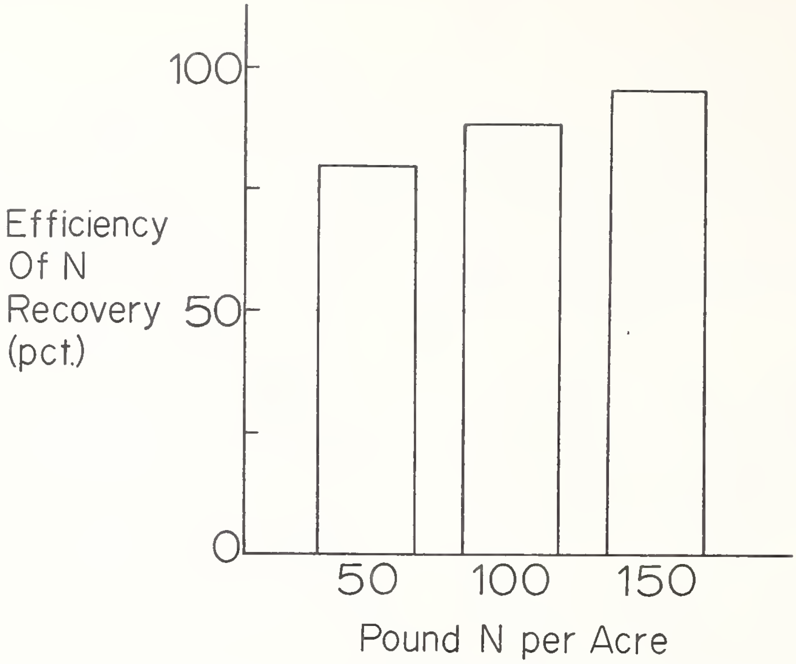

Figure 7. Nitrogen efficiency in producing protein in an orchardgrass — blue grass sod. Lexington, 1971

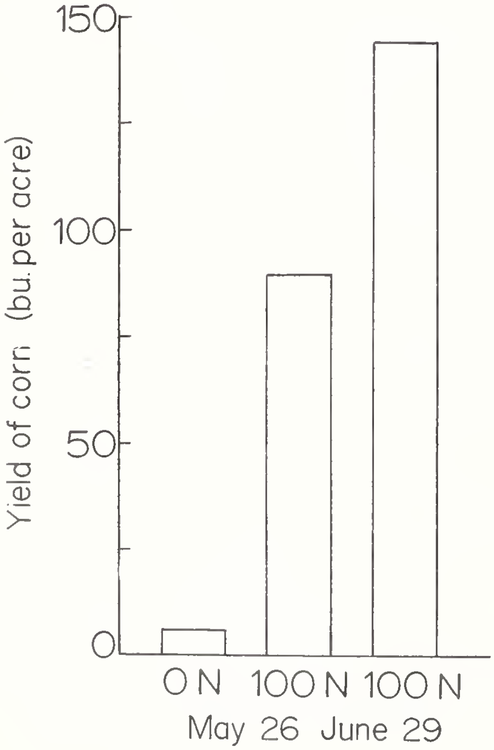

Figure 8. Effect of nitrogen fertilizer application date on corn yield. Melvin soil, 1971, Hartford, Ky.

Fertilization

Fertilization is simply a means of increasing the nutrient content of a soil during plant growth so that the plants can live up to their genetic potential for yield. In the case of nitrogen, as we have indicated, the objective is to have a sufficient quantity present during the period of crop need. This is a short period for some crops such as corn and a longer one for crops such as grass. Since this is the objective, the best way to meet it is to apply nitrogen just before it is needed. This makes sense from both economical and ecological points of view. In Kentucky, we have worked for several years on this problem with corn. Some results are shown in figure 8. On this soil, where denitrification and leaching of nitrate are problems, the most efficient time to add nitrogen fertilizer is about a month after planting. Some soils have much more flexibility than this, but many of our wetter bottomlands fit this picture rather well.

The amounts of nitrogen that should be used for economical but nonpolluting yield are hard to determine. They do vary considerably from one soil to another. Unfortunately, they also vary from one year to another. We can predict soil behavior much more accurately than we can predict weather behavior, however. The result is that we make recommendations based on known soil conditions but based "average" weather. This results in applications that are too low in wet years and too high in dry years. It also results in some economic loss to farmers and some contamination of water.

Mineralization of Nitrogen

In most soils, the mineralization of nitrogen from organic matter and its conversion to nitrate constitutes an important source of nitrogen for plants. The breakdown in organic matter is influenced by oxygen and water contents of the soil and by soil temperature. Under optimum aeration and moisture and with a rising temperature, the mineralization of organic nitrogen to nitrate proceeds most rapidly. This occurs in the spring at about the same time that fertilizer is applied. In our best soils, the nitrate-nitrogen formed in this way may, in fact, exceed 100 pounds per acre, about equal to the fertilizer nitrogen added for corn. This mineralization on a nationwide scale, completely dwarfs the application of fertilizer nitrogen because it happens on all land to some extent — not just on cropland. An example of the speed of nitrate formation under ideal conditions is shown in figure 9. Many soils do not produce nitrate at this rate, but our better cropland does. This "natural" nitrate can enter water just as surely as fertilizer nitrate can, and our organic gardening friends should be made aware of that fact.

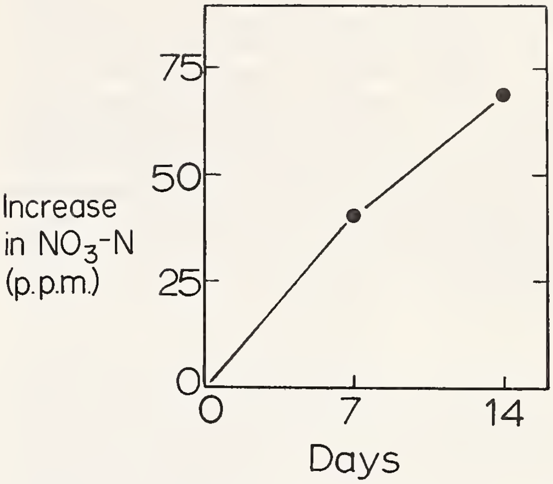

Figure 9. Mineralization of nitrate-nitrogen during a 14-day period. Maury soil, Lexington, Ky. spring, 1970.

Nitrate-Nitrogen Levels of Streams

In 1971, we began to sample streams flowing from several agricultural watersheds in Kentucky. Some results for 1971 are shown in figure 10. The watersheds are located in all regions of Kentucky. This probably occurred because the

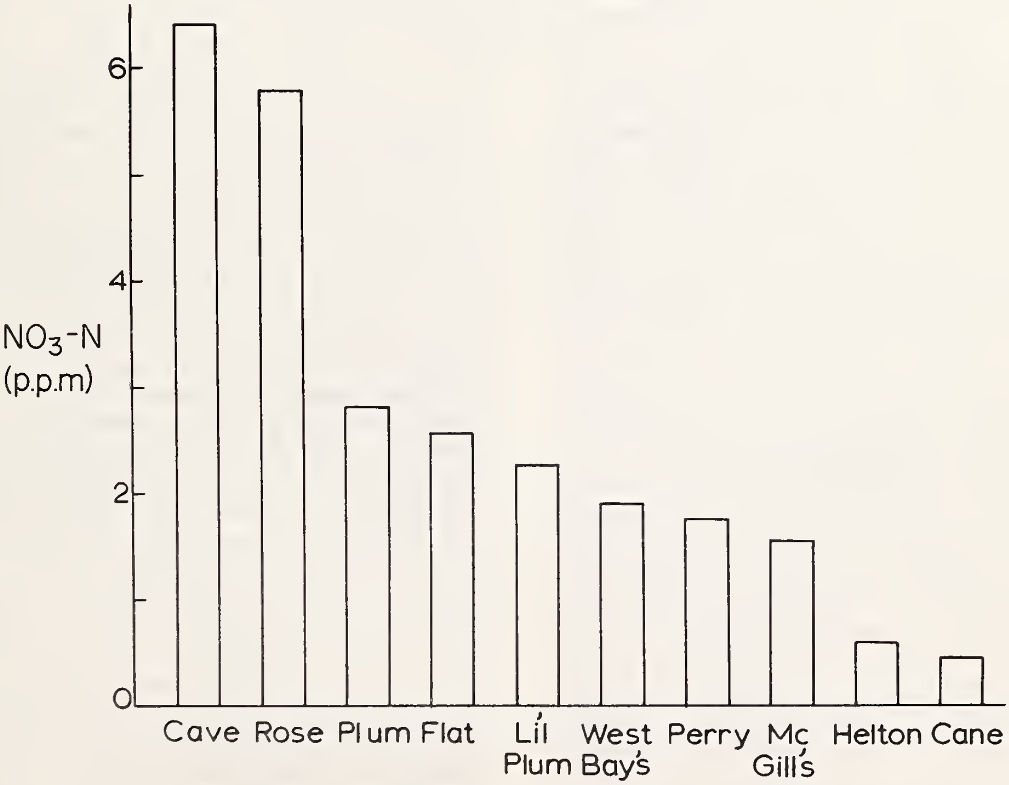

Figure 10. Nitrate-nitrogen levels found in 10 Kentucky streams draining agricultural watersheds, 1971

soils are high in organic matter and very fertile. Mineralization, much more than fertilization, was the cause since little fertilization is used. Rose Creek was second highest and is a heavily cropped watershed. Fertilization of corn probably contributed substantially to the level observed. Helton Branch was the lowest and is essentially all in woods. This represents an ideal situation in the minds of many people. They do not stop to wonder why the watershed might be all in woods, however. The other watersheds show intermediate values and are in mixtures of grass and woods except for Perry Creek which is heavily cropped. Obviously, no simple pattern emerges.

Conclusions

As a result of the watershed data and the many uncertainties associated with the reactions of nitrogen in soils, it is not possible to say with much accuracy what the nitrate level of water will be in a given area. However, it is possible to make some conclusions about things that affect the nitrate level of ground and surface waters. A list of these follows:

1. It is economically and ecologically unsound to add a great excess of nitrogen over what the plant requires.
2. Nitrogen should be added to soils as closely as possible to the time when plants require it.
3. On red soils more carelessness in time and rate of nitrogen can be allowed.
4. In soils where denitrification is a severe problem, extra nitrogen must be added to assure an adequate crop, but contamination of water is less likely.
5. Mineralized nitrogen from soil organic matter or added organic matter contaminates water just as fertilizer nitrogen does.
6. Anything that reduces evaporation from soil will increase leaching of nitrate.

SESSION II: "Control of Health Problems" — Jay C. Murray, Agricultural Experiment Station, Oklahoma State University, Stillwater, Chairman

BENEFITS OF NUTRITION RESEARCH ON HEALTH

by

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Better health, a longer lifespan, and greater satisfaction from work, family, and leisure time are among the benefits to be obtained from improved diets and nutrition. These are the traditional reasons for undertaking and supporting research in the varied aspects of human nutrition. Until recently, it has been enough to speak in these general terms. During the 1960's, there was increasing pressure to translate the benefits into dollar or economic terms to provide a common basis on which administrators and supporters of research could evaluate alternative research proposals both within the nutrition area and in relation to other research needs.

Attempts to establish an economic basis for evaluating research or to develop cost benefit data were made in industry as early as the 1930's. However, no satisfactory formula has been devised for applying the cost benefit system to basic research, unless the basic research is closely related to the applied problem. Since the introduction of the Program-Planning-Budgeting system in the Department of Defense in the early 60's, considerable thought within the Federal establishment has been given to procedures for deriving cost-benefits for all types of research. Several efforts have been made to establish economic benefits for human nutrition research, both in the United States and in the United Kingdom.

About 3 years ago, the U.S. Department of Agriculture (USDA) and the State Universities and Land Grant Colleges undertook an evaluation of human nutrition research in the United States. A part of the study dealt with the benefits that could be realized from improved diets and nutrition. Estimates were made of the potential savings. These are value judgments based on review of scientific literature, health statistics, and discussions with clinicians. No direct evidence on which to base the judgments exists. The study was intended to provide a base for future discussions, theory development, input of divergent viewpoints, and updating. In fact, the estimates in the USDA study may be too low on the basis of evaluations since published by other knowledgeable groups.

The long-range goal of most human nutrition research is dietary improvement so that optimal health, well-being, and satisfaction may be ensured or alternatively, the prevention, modification, or postponement of health problems and of the aging process. Most nutritionists and clinicians feel that the real potential from improved diet is preventative. Proper diet may defer and modify the development of the disease so that the clinical condition does not develop. Who would benefit most from improved nutrition? The answer is everyone would share. Benefits would be available to the entire population. Each age, sex, ethnic, economic, and geographic segment would be benefited. The lower economic and nonwhite population probably would benefit most from the effective application of current knowledge. However, savings to these special groups comprise only a small part of what might be accomplished for the entire population from future research. Some of the advantages can be expressed as dollar benefits to individuals, families, communities, industry. or to the Nation. The social and personal benefits are harder to quantify and describe. It is difficult to place a dollar value on the avoidance of pain or the loss of a family member; satisfactions from healthy, emotionally adjusted families; career achievement; or the opportunity to enjoy leisure time.

In characterizing the benefits, three general factors were considered. The first was who benefits. Was a particular population group involved? What age, sex, economic, ethnic, and geographic population group? Is the benefit realized by the individual, the family, an economic segment, or the Nation as a whole? Secondly, in what manner is the benefit realized? Is it through better health, improved individual performance and satisfaction, or increased efficiency in food services? The third aspect is how can the benefit be quantified. This can be done in a number of ways. For instance, the number of persons involved, work or school days lost from disability, medical and hospital costs, loss of life, longer lifespan, delay in aging resulting in a longer productive lifespan, reduced accident rates, improved work efficiency, and more efficient use of existing food supplies and services. A great deal of attention is currently being given to estimating the savings which may result from improved work efficiency, growth, development, and learning ability as a result of better nutrition. Not enough is known of the relationship of nutrition to these conditions to provide an objective measure of most of these benefits. At this time, it is possible only to make value judgments. This basis for evaluating nutrition benefits is of great current interest, both in the United States and elsewhere.

The types of benefits from human nutrition research may be considered under three major groupings: (1) The prevention, amelioration, and postponement of nutrition-related health problems, (2) improved individual performances and satisfactions, and (3) increased efficiency in food services.

The greatest impact would be through improved health. Most all of the major health problems in the United States are diet related. A consideration of the leading causes of death in 1969 suggests that all of the listed problems could be modified by improvements in nutrition (fig. 1). By far, the leading cause of death is diseases of the heart followed by cancer, vascular lesions attacking the central nervous system, accidents, influenza and pneumonia, certain diseases of early infancy, diabetes mellitus, arteriosclerosis, other bronchopulmonic diseases, and cirrhosis of the liver. Expenditures for health care in the United States are skyrocketing, accounting for 67.2 billion dollars in 1970 or 7.0 percent of the entire U.S. gross national product. Death rates for many of these conditions are higher in the United States than in other countries of comparable economic development.

Major health benefits are long range, and for this reason are diffcult to identify with specific diet changes. The problems that are now facing nutritionists are not those which respond dramatically and immediately to diet change. Predictions of the extent to which diet may be involved in the development of various health problems have been based on the current knowledge of metabolic pathways typical of persons in advanced stages of disease. There is little understanding of when or why these metabolic changes began. The human body is a complex and very adaptive mechanism. For most essential metabolic processes, other pathways exist which can be utilized in response to physiological, diet, or other stress. Frequently, a series of adjustments take place and the end result does not become apparent for a long time, even years, when a metabolite such as cholesterol accumulates. Early adjustment of diet could prevent the development of undesirable long-range effects. Minor changes in diet and food habits instituted at an early age might well avoid the need for major changes difficult to adopt later in life.

Examination of vital statistics for the United States has some provocative implications for diet-health relationships. For instance, there are some distinct regional differences in death rates and health problems which are likely due to environmental effects. While the evidence is circumstantial, it is very likely that food habits, food composition, and water content contribute to these regional differences. The lowest death rates in 1959 to 1961 for white males age 45 to 64 were scattered through the Midwestern States, in Arkansas, and one location in Tennessee (fig. 2). The highest death rates were along the eastern seaboard, one location in Pennsylvania, and a wide area in Nevada. The regional distribution for women the same age is a little

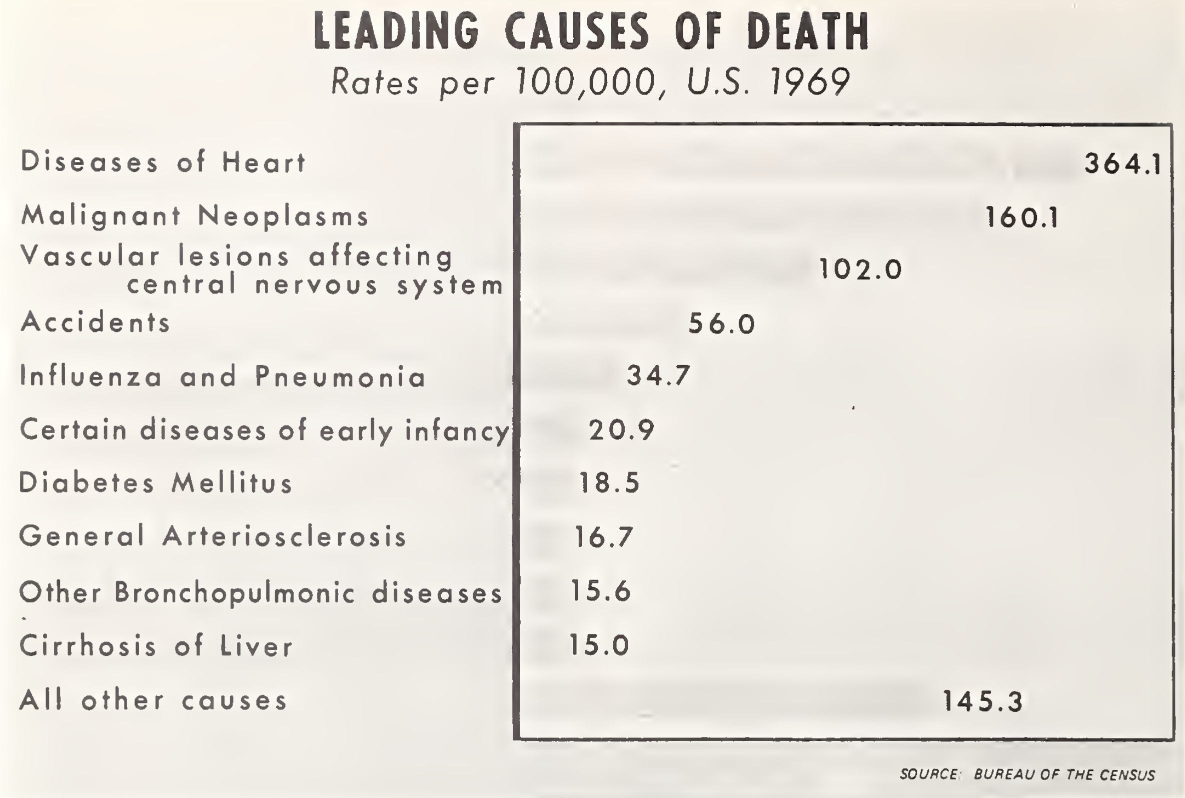

Figure 1. Leading causes of death. Rates per 100,000, U.S. 1969

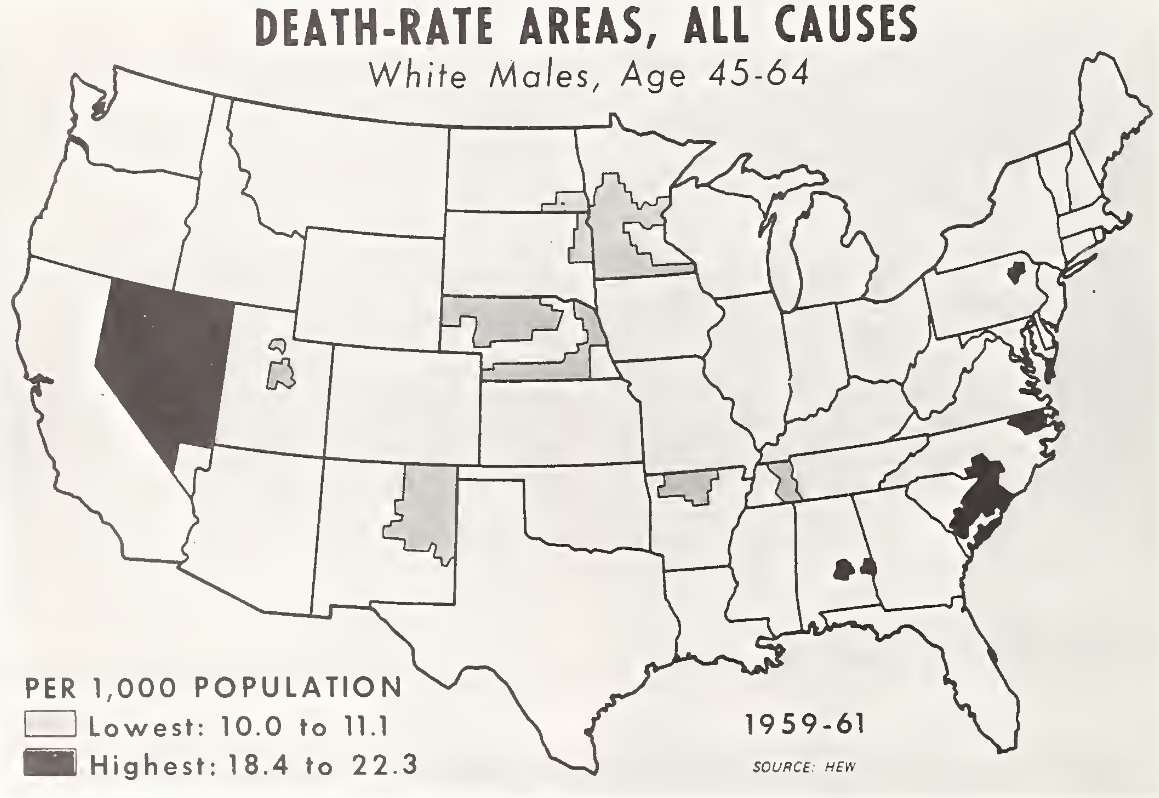

Figure 2. Death-rate areas, all causes, white males, ages 45-64

different. High death rate areas are more concentrated in the Pennsylvania and Delaware region in the east and upper Michigan. Low death rate areas are in the Midwest but more towards the South. Death rates for the women in this age group were about one-half that for men with the highest death rates, about comparable to the lowest for men.

It is generally accepted that the food distribution system in the United States has removed, for all practical purposes, differences in nutrient intake which may be due to local food production. However, a part of the food is grown and processed locally. Processing procedures are often adapted to meet local food likes and dislikes. In addition, these differences in death rate may reflect the accumulative effect of chronic low intake levels of some nutrients throughout the lifespan and by successive generations. A number of examples of regional health problems attributable to differences in the nutrient content of food or to dietary pattern can be given. For example, in the "goiter belt" where soil and plants are low in iodine, the high incidence in death rate of goiter was reduced when the diet was supplemented with iodine. Demographic studies reveal that human breast cancer incidence is also high in iodine-deficient areas.

Migration from the high death rate areas almost always results in a reduction in death rate, although the improvement never approaches the level achieved by those who were born and continue to live in low rate areas. Similarly, persons who move from the low rate areas into higher rate areas lose part of the advantage. If the death rate for one of the high death rate areas, Wilkes Barre, Pa., were applied to the entire U. S. population, 140,489 more persons under 65 years would have died per year during the period 1959 to 1961. If the death rate for one of the lower rate areas, Nebraska, had prevailed, there would have been 131,634 fewer deaths. These findings suggest the magnitude of benefits that would be realized if all areas had the same low death rates as Nebraska. The highest death rate areas generally correspond to those where agriculturists have recognized the soil as being depleted for several years. This suggests a possible relationship between submarginal diets and health of succeeding generations.

There is a great deal of epidemiological data suggesting that diet plays a major role in the development of heart disease, the number one cause of death in the United States. Elevated blood lipids and cholesterol, reduction in glucose tolerance, and obesity are all high risk factors in cardiovascular diseases. All are known to respond to diet changes. The likelihood of death from arteriosclerotic and degenerative heart disease is much higher in the United States than in many other countries in the western world. The United States ranks sixteenth. There is also a marked difference in death rates from heart disease within the United States corresponding generally to those for all causes of death. Any consideration of the reasons underlying geographic differences in death rate must include diet. What foods are eaten? Do the foods differ in nutrient content or value among the areas? What differences exist in the mineral content of the water supply? There is evidence of a lesser development of atherosclerotic lesions with high calcium intakes. It also has been suggested that there is a lower incidence of coronary heart disease in areas where the water is hard.

The importance of diet compared with the other risk factors is not known. Substantive data have not been obtained on the role of diet before or during the development of cardiovascular problems. Most diet studies have been made of individuals who have already had one coronary incident. There is no proof, but considerable evidence, that to be effective, any changes in dietary patterns should begin at an early age in order to delay the onset of these diseases. The President's Commission on Heart Disease, Cancer, and Stroke in 1964 estimated the cost of heart and vasculatory disease to the Nation at over 5 million people in 1962 with definite or suspect heart disease and an economic cost of 31.6 billion dollars. The potential savings from improved diet in this one health area alone would be enormous if nutrition could effect a 25-percent reduction in the number of people with heart disease under age 65 and a 20-percent reduction in the economic costs. This is considered a reasonable estimate by most specialists in diet-heart relationships. In January 1972, Dr. Cooper, Director of the National Institute of Heart and Lung Disease, reported that a committee of that institute estimated a 90 percent reduction in deaths from arteriosclerosis could be achieved if Americans cooperated by modifying their diets and cutting out cigarettes.

Respiratory and infectious diseases are another health area where diet may play an important role and affect great savings. Acute respiratory infections remain the most frequent cause of illness and the most important cause of loss of time from work and school in the United States. One-third to one-half of industrial absenteeism from sickness is caused by acute respiratory infections. In addition, mild infections may reduce efficiency without occasioning absenteeism. The economic importance of morbidity from acute respiratory infections is impossible to determine precisely, but it has been estimated to be well over 5 million dollars per year. In addition, 1 billion dollars alone are spent for cold remedies and facial tissues. In 1969, there were 110 acute respiratory and 25 infective medically attended cases per 100 population.

Diet and the nutritional state of the individual involved are clearly associated with the incidence, duration, and severity of respiratory and infectious diseases. There are few statistics available to show the direct relationship between nutrition and infection. There is, however, evidence that body stores of nutrients may be reduced severely by acute infections. For example, in children many acute infections reduce the levels of Vitamin A in the blood as does vaccination against smallpox and measles. Xerophthalmia, night blindness, frequently follows these infections, indicating the depletion of body reserves of vitamin A. Vitamins B_1, B_6, and C, and protein also are implicated. There are no satisfactory ways to determine the extent of body stores for most nutrients or to identify the level of nutrient intake needed to maintain adequate stores for resistance to infection. Respiratory and infectious diseases caused a loss in 1965 of 141 million work days and 166 million school days in addition to medical and hospital costs. Perhaps a 15 to 20 percent reduction could be made by maintenance of adequate nutrient stores. Work days lost mean an economic loss to the employer and may also result in salary loss for the individual. School days lost are more difficult to evaluate because lost opportunities to learn may never be made up.

The potential improvement in mental and emotional health which might be brought about by improved diets is an unknown but exciting possibility. Information to substantiate a direct relationship between diet and mental and emotional health does not exist. There is no doubt that mental disorders can lead to poor eating habits and malnutrition. Also, it is clearly established that good nutrition is necessary for proper development and functioning of the central nervous system. Dietary improvement results in increased resistance to infection, better management of alcoholics, fewer circulatory disturbances in cardiovascular conditions, control of metabolic disturbances, diabetes, and hyperthyroidism, and fewer nutrient deficiencies, all of which have been associated with mental disabilities. Conceivably, as much as 80 percent of the U.S. population could benefit from improved mental health with 12 percent having a major benefit. Benefits would be economic through reduced hospital and psychiatric costs, improved ability and opportunities on the job, and fewer work days lost. Social benefits would be of even greater importance as represented by less family stress, fewer broken homes, and greater social acceptance of the individuals and their families. The potential benefits in terms of mental health from improved diet could be enormous and may well prove to be a major advance made possible by the recently developed procedures for the study and manipulation of prenatal nutrition.

During this century, the infant mortality rate in the United States has shown a constant decline, but it has not declined as rapidly, or to as low a level as in several other countries. In 1950, the United States ranked sixth among a group of countries with characteristics which make comparison possible (fig. 3). In 1961 to 1963, the United States had fallen to 10th place, and to 13th in 1968. Sweden had the lowest average annual infant mortality rate in 1968, about 13 per thousand. The 1968 infant rate in the United States was 21.7, a slight decrease from 22.4 in 1967[1]. In that year, the mortality rate for white infants was 19.7 per thousand as compared with 35.5 in nonwhite infants. There are several causative factors for these differences in infant mortality rates, and one of them is diet. The incidence of low birth weight in the United States is steadily increasing, as is the percentage of infant deaths due to birth defects. Both of these changes could be diet related. Many millions of children have handicaps. In fact, the 1964 vital statistics survey in the United States showed the congenital defects including genetic metabolic disorders was the leading cause of death in the first year. Actually, as a cause of death, birth defects are outranked only by heart disease.

Malnourishment in the mother usually results in the birth of a baby who is underweight. These babies are more likely to have birth defects. This has particular significance in the United States where there are probably more child pregnancies than in any other Nation in the world. In 1965 in the United States, there were more than 196,000 live births to girls 17 years of age or younger. These young mothers are in the sex-age group most likely to have nutritional deficiencies as indicated by the National Nutrition Survey in 1968-69 and the data for individuals obtained during the Nationwide Household Food Consumption Survey of 1965.

Another area where nutrition plays a major role is in the prevention of early aging and an increase in active lifespan. Aging is an inevitable and irreversible process which begins no later than early adulthood. Improved diet has as a goal the deferment of the aging process, so that people can benefit from better health, less pain,

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[1] Provisional figures for 1970 were: Sweden — 11 deaths per thousand; United States — 19.8 deaths per thousand.

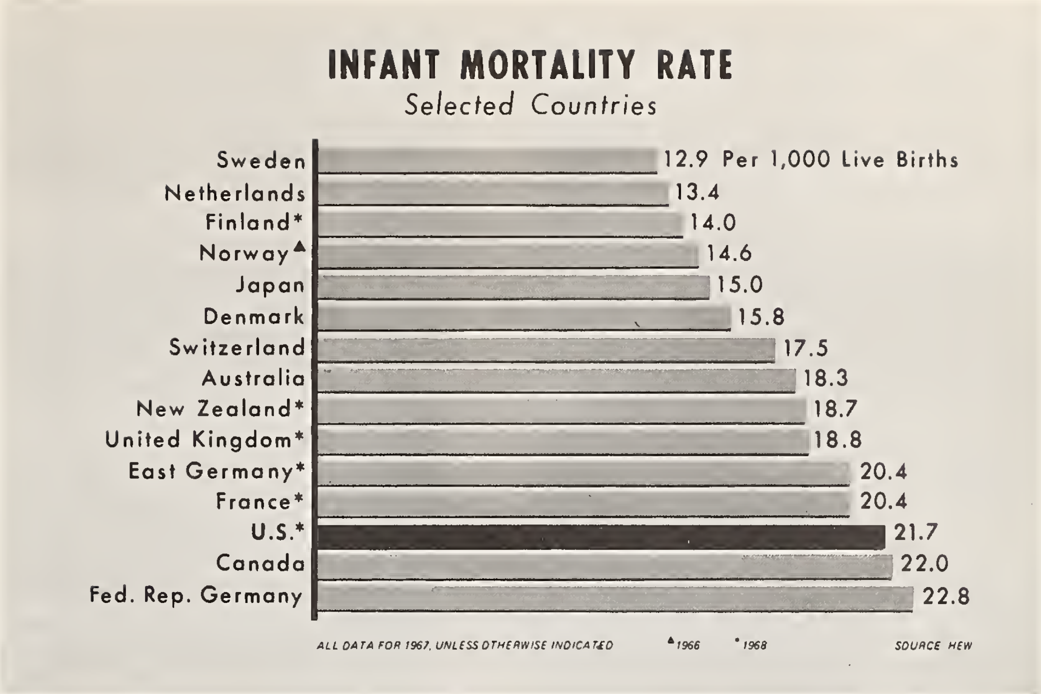

Figure 3. Infant mortality rate of selected countries

increased activity, and satisfaction throughout life. The impact of aging changes becomes more obvious and important as man increases his ability to control infectious diseases, trauma, nutrition, and environmental stress. Expectation of longer life improved as did the proportion of men and women surviving to adulthood and older ages. Similar changes took place in most countries but some have plateaued at a lower rate than the United States; for instance, Denmark has a death rate of about 7.5 per hundred thousand compared with 9.3 in the United States. The mean lifespan in the United States has remained essentially constant since 1950 (fig. 4). The rate of increase in mean lifespan in the future is likely to be slow unless new ways are found to inhibit biological degradation.

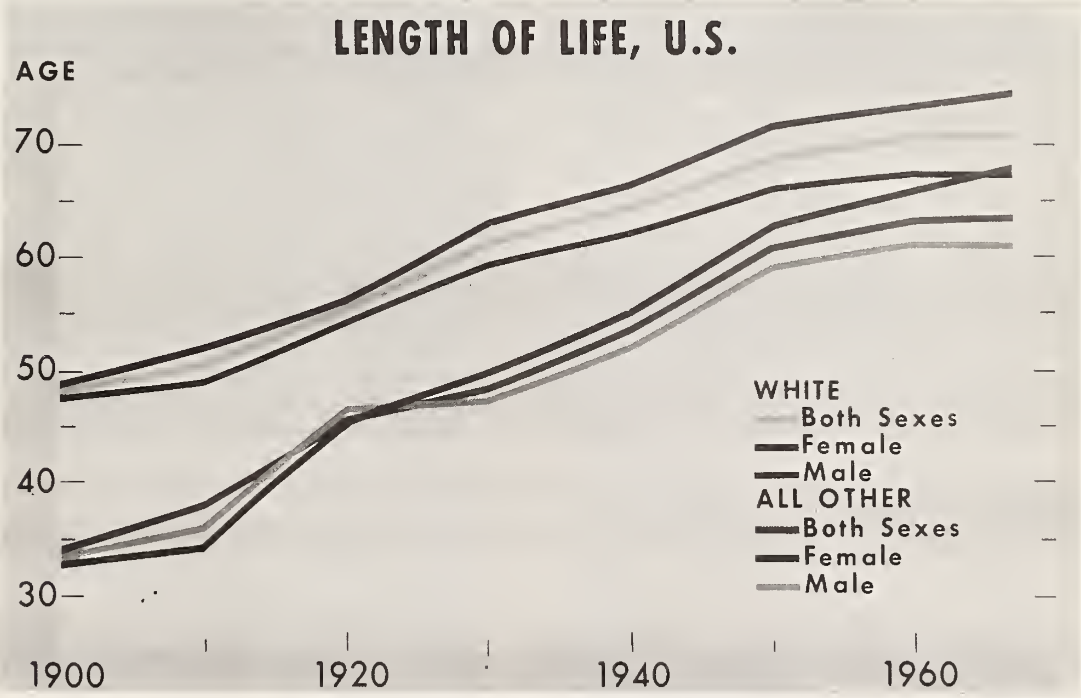

Figure 4. Length of life, United States

SOURCE: HEW

The time between nutritional insult and measurable effect in terms of health may be quite long. A period of 40 or more years may elapse before the clinical symptom becomes visible. For example, most persons over 40 years of age metabolize glucose less efficiently than young persons. This impairment in carbohydrate metabolism may have an important bearing on the development of atherosclerosis. It has been suggested that an underlying cause of the decrease in ability to metabolize glucose is a chronic marginal deficiency of chromium in the diet. Chromium is one of the trace elements in food which has been shown within the past fews years to be essential. This mineral, particularly its biologically active form, occurs in very small amounts in food. Nutritionists have had small concern for the trace minerals because it was generally thought that these minerals were needed in such small amounts and were so widely distributed in food that dietary intakes would be adequate without special attention. Advances in food processing and the fabrication of foods has made their dietary intake critical.

Benefits from slowing the aging process and increasing lifespan are difficult to quantify in an economic sense. These measures of benefits are suggested: A reduction in the number of people having one or more chronic impairments from 102 million to 92 million persons; a gradual improvement in the number of people surviving to age 65 up to 90 percent. The present survival to age 65 is: For white males, 66 percent; for Negro males, 50 percent; for white females, 81 percent; for Negro females, 64 percent. No goal was set for increasing life expectancy except to bring the expectancy for Negroes up to the same level as that for whites.

Benefits were proposed for a number of other health problems. For instance, arthritis and rheumatic diseases cause a greater loss of time from work than any other set of health problems, with the exception of nervous and mental disorders and respiratory diseases. The loss was recently estimated at 27 million days annually. About 16 million people in the United States are affected. About one of four victims is restricted from carrying on any major activities and one of ten is confined to the home. To control the progress of this disease, it is important to enhance protein and calcium ingestion. Conceivably, if we knew enough about the nutrition and dietary needs of people, the incidence and severity of arthritis might be modified. Possibly, the number of people afflicted and work days lost might be reduced in half. The potential savings thus from improved diet would include 8 million people who would not have the affliction or would have it in mild form which would not be debilitating; 13.5 million work days saved; 125,000 more people employed; and an annual dollar saving of 900 million per year.

This type of rationale has been applied to several other health problems. For instance, dental health, where it is suggested that a 50 percent reduction in incidence, severity, and expenditures may be brought about by improved diet. Incidentally, one-half of all people over 55 have no teeth. Diabetes and carbohydrate disorders affect many people. Seventy-nine percent of people over 55 years have impaired glucose tolerance. Conceivably, diet could improve 50 percent of these cases. Osteoporosis affects 25 percent of women over 40 years of age, and there are 8 million severe cases. While this report was being written, data were published suggesting that osteoporosis could be the result of a prolonged chronic submarginal intake of calcium. The investigators were able to reverse some of the changes typical of osteoporosis by increasing the calcium intake. Thus, an increase in the calcium intake of older persons might control the progressive development of a disease which is one of the major causes of disability in old age. An optimistic estimate, perhaps as much as 75 percent reduction in disabilities from osteoporosis, may be claimed.

Obesity, while not itself a cause of death, is a high risk factor for many health problems. Death rates of overweight men are higher than expected from general population estimates. Obesity is found throughout the population with 3 million adolescents being affected, 30 to 40 percent of all adults, and 60 to 70 percent of those over 40 years of age. Improved diets and nutrition made possible by better knowledge of metabolic processes, food requirements, and factors motivating the establishment and maintenance of food habits might reduce the incidence of obesity by 80 percent. A number of other health problems have been similarly evaluated, including alcoholism, eyesight, hair, skin and nail problems, allergies, digestive diseases, kidney and urinary diseases, and muscular disorders.

The potential role of diet in cancer is worthy of note because, here again, the next few years may bring a clarification of our present knowledge. The incidence of deaths has steadily decreased since 1930 for cancer of the stomach and uterus and increased for lung cancer and leukemia. The incidence and deaths for all types of cancer increases with age. Many cancers are believed due to viruses. There is a small, but growing, body of data suggesting that chronic low level intake of some nutrients is a factor in the incidence of cancer in man. There is evidence that vitamin B deficiency plays a role in the occurrence of cancer of the oral cavity in the esophagus. Leukemia cells in children require both more folic acid and more asparagine. Chronic vitamin B deficiency, due to an inadequate supply of vegetables in the diet, appears to be incriminated.

Data relating nutrition to the incidence and control of cancer are still too fragmentary and hypothetical to provide a basis for estimating benefits. The results of ongoing research may justify appreciable benefit claims for regulation of diet in the avoidance and management of cancer. In 1968, 600 thousand people developed cancer and 320 thousand persons died of cancer.

Improvements as a result of relieving known dietary deficiencies would appear a simple area for which to express benefits. On the contrary, no benefits have been identified or quantified. The reason for this is that the effects of vitamin deficiencies at any age, particularly in young children, are reflected both in greater likelihood of immediate health problems and of health problems in adulthood. For instance, vitamin A deficiency in young children may well be a contributing cause to problems of eyesight in adults. Nutrient deficiencies also reduce work efficiency, limit growth, muscular and nervous development, and interfere with learning ability. All of these have lifetime effects. The National Nutrition Survey and the Nationwide Household Food Consumption Survey both indicate that the young and the poor are most likely to have inadequate nutrition. Examination of the vital statistics in the United States invariably show that the nonwhite population and the poor have higher incidences of both mortality and morbidity.

One of the most challenging and difficult tasks in delineating benefits from improved diet comes in defining the benefits from better growth and development and personal satisfactions. A close reltationship between nutrition, growth rate, and health has been recognized for years. Increases in heights and weights of children in proportion to age has not increased much in the United States in recent years. From this, it might be inferred that the genetic potential for growth of most Americans has now been reached. However, there are still children from low income families who are retarded in growth as shown by data from the recent National Nutrition Survey. Height rather than weight is affected in these children, and it is not expected that this early growth retardation in preschool age children can be reversed by diet at a later age.

There is a good deal of evidence with animals and some is accumulating with children that improved diet may improve learning ability. It may play a role in the incidence of mental retardation when the malnourishment occurs in the critical months before birth and shortly after. In addition, malnutrition in school age children and adults may handicap learning ability by shortening attention span and decreasing the ability to work at the same task for any sustained period.

Current experiences with the school breakfast, school lunch, and preschool feeding programs should provide some substantive data. There remains, of course, the need to quantify the extent to which learning ability and working ability in the United States is affected by diet. This is more readily done in countries where a large proportion of the population is engaged in physical labor. Even there, no satisfactory means of expressing these benefits in economic terms have been developed. Innovative approaches will be required. An increased interest in developing such figures is needed.

For the families where income is not a limiting factor, there is also a problem concerning growth and development. Very little is known of the long range implications for man of rapid growth during infancy and childhood. It has not yet been demonstrated in man, but long term studies with rats have shown that those animals that reach their adult weight early have a markedly reduced total lifespan. It is difficult to arrive at the ideal growth rate and the criteria for its establishment. As long as adequate growth and development are achieved for the individual to cope with the tasks faced, what benefit is to be gained from his growing 1 to 6 inches higher? To what extent does it increase his efficiency in working and how important is this in a society where technology is developed and work output is governed by economic factors? True, malnutrition as well as undernutrition decreases the worker's productivity. Apathy, lethargy, and lack of initiative result. Ability to learn new tasks may be reduced. The resistance to disease also is lowered, increasing the rate of absenteeism from the job. Thus, in a number of indirect ways improved diet may increase working efficiency due to feeling better on the job and so lead to economic benefits. A 0.5 percent increase in on-the-job efficiency has been suggested as a possibility from improved nutrition. The benefit when expressed in terms of increased wage and salary dispersements over the total in 1968 approximated 2.3 billion dollars. If even 20 percent of this benefit were achieved, the savings would be significant.

Accident rates are high among those who tire quickly due to malnutrition. In 1967 in the United States, accidents ranked fourth as a cause of death, accounting for over 113,000 deaths. In addition, 51.8 million people were injured severely enough to restrict activity or require medical attention. These accounted for 324.5 million days of disability. Accidents also accounted for almost 10½ million lost school days and over 88½ million days lost from work.

This discussion has dealt mainly with benefits from improved health, working, and learning ability of individuals. Other mainly economic benefits from human nutrition research also may be claimed. Benefits from improved efficiency in food preparation may be expressed in terms of time, labor, and cost savings in the home and service institutions. Mechanization and automation of some aspects of food preparation may be more economically carried out only for large quantities of food. Byproducts of food preparation which would be considered waste in the home or institution may be transformed to useful products when available in larger quantities in a commercial situation.

Improved food preparation techniques would reduce the loss of some nutrients and increase the availability of others. Losses up to 90 percent of water soluble, heat sensitive vitamins may occur when cooked foods are held for long periods of time or are reheated, both common procedures in the use of prepared foods. Much of this loss occurs before the reheating process. Processors' attention is centered on palatability, not nutritive value, when prepared food products are designed and methods for reheating are recommended. New processing techniques coupled with improved methods for handling and preparing processed foods could ensure vitamin losses no greater than 25 percent.

The final area considered in evaluating benefits from improved diets was improved efficiency in food programs. Maximum improvements in diets and nutrition can be achieved only through coordinated planning by nutritionists, food technologists, economists, and educators. Coordinated planning by all of the disciplines is needed to ensure that food programs accomplish the goal of wiping out malnutrition in the shortest possible time and at a minimum cost. The goal should not be limited to doing away with undernutrition but should include reducing malnutrition regardless of income level. Programs should encompass educating people to make proper selection of food; making sure that the foods available to consumers contain the necessary nutrients for needs to be met through the normal food chain; and to ensure that no one, for economic reasons, lacks the foods necessary for proper diet. The profits from such programs will be seen in improved health; well-being, and satisfaction of everyone.

ACKNOWLEDGEMENT

This paper is based on a report "Benefits from Human Nutrition Research," issued August 1971 by the Science and Education Staff, U.S. Department of Agriculture, Washington, D.C. This report is part of a study conducted at the direction of the Agricultural Research Policy Advisory Committee, U.S. Department of Agriculture. A joint task group representing the State Agricultural Experiment Stations and the U.S. Department of Agriculture was assigned the responsibility for making the study.

FOOD FATS AND HEALTH

by
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The United States Department of Agriculture (USDA) has for decades been concerned with diets which can maintain healthy and well nourished populations. The USDA also has the responsibility that the food produced by this country supply these needs. It is equally important therefore that we be aware of any needed changes to meet new dietary demands. To achieve these ends, nutritional research and technological improvements in food production must move together to further our knowledge for planning and evaluating programs for improving the health of the Nation.

Factors Complicating the Role of Diet and Heart Disease

Nutrition has been shown to be an important factor of our environment that can influence the development and degree of heart and vascular disease. Over half the deaths in the United States at the present time are related to some form of cardiovascular disease. In addition, untold numbers of cases of morbidity and disability in various stages of this disease afflict man, resulting in enormous health problems.

The most prevalent form of cardiovascular disease is atheroschlerosis, which is characterized by the accumulation of fatty material in the walls of medium and large arteries. Both genetic and environmental factors have been implicated in the etiology of this disease. Considering the magnitude of the problem, it is evident that a major factor involved in the development of atherosclerosis is the overwhelming effect of the environment. Environmental factors, such as diet, smoking, obesity, psychosocial tensions, sedentary living, and high blood pressure, all combine to influence the development of overt symptoms.

In the USDA, we are concerned with diet and its relationship to health in man. Generally speaking, experts in the field of diet and heart disease agree that the fat content of the American diet should be moderately decreased.

Before major changes are made in the diets of the general population, confirming evidence is needed from experimental studies for estimating the acceptable levels at which foods such as meat, dairy products, and poultry be eaten. A careful assessment must be made to determine (1) to what degree blood lipids are lowered, (2) how blood clotting parameters, particularly those related to thrombosis, are improved, and (3) to what degree the regression of atherosclerosis occurs with reduced fat intake. In order to answer these questions, long-term dietary studies in man will have to be undertaken, where great care is taken to avoid complicating the interpretation of nutritional studies with other environmental factors that can also influence the development of this disease. It is important, also, that our knowledge of dietary sources of lipids be carefully re-evaluated in the light of production and processing techniques that alter the structural characteristics of lipids.

Elevated fat patterns of blood continue to be associated with cardiovascular disease, the leading cause of death. The most serious attempts to reduce blood lipids in man thus far have been through the substitution of polyunsaturated fats for the saturated fats. An evaluation of the many nutritional studies in man reveals that an average decrease of about 10 percent in blood cholesterol has been achieved by this means. Such a reduction in blood cholesterol is not considered sufficient to bring man into a "safer" zone. Moreover, there are a number of unanswered questions as to the side effects of feeding high levels of polyunsaturated fat. From a dietary point of view, a currently promising alternative is to moderately reduce the saturated fat-cholesterol content of the diet.

Other nutritional factors and interrelationships are implicated in the regulation of lipid metabolism. For example, it is yet to be determined if calories from carbohydrate, especially the simple type sugars, if used to replace calories from fat, have a similar metabolic effect to that of calories derived from fat.

The role of dietary calcium in reducing blood lipids is not clearly understood, nor is the role of other trace mineral elements such as magnesium, vanadium, chromium, copper, and zinc. B-vitamins such as biotin, pyridoxine, and nicotinic acid have also been shown to alter fat patterns of the body. Very little attention has been given to the levels at which these micronutrients should be eaten in order to avoid abnormal blood lipid patterns.

Interim Suggestions for Diet Changes

Provisional recommendations for dietary fat are being made in the light of current knowledge of human nutrition and for the purpose of reducing the incidence of morbidity and mortality in the population as a result of widespread cardiovascular disease in our country. The suggestion is intended as an interim measure because existing research findings are inconclusive. Interpretation of information coming from the scientific literature is still controversial and in many instances scanty. It is still risky to make dietary recommendations for the population as a whole for fat calories of the diet, kinds of fat to be eaten, and the level of cholesterol.

We are being guided by the dietary fat recommendations of the report of the Inter-Society Commission for Heart Disease Resources with minor modifications. The proposed reduction of total fat intake of the American diet has necessitated the re-evaluation of the whole diet to make sure it can meet the total requirements of individuals in all age categories and of both sexes.

Although much basic information is still needed in relating dietary fat to health, tentative, interim recommendations are needed because of the current health problem. Keeping these provisions in mind, the following nutritional recommendations are made:

1. Caloric intake to achieve and maintain optimal weight.
2. Reduction of dietary fats, particularly saturated fats. It is recommended that the total fat intake of the diet be reduced to 35 percent of calories or less, and that the saturated, monounsaturated, and the polyunsaturated fats be included in the diet in approximately a 1:1:1 ratio.
3. Reduction of dietary cholesterol to 500 milligrams per day or less.

These recommendations are approximations. It is further recommended that blood cholesterol be maintained at 200 mg. percent or less. If this level of cholesterol cannot be maintained by the dietary fat recommendations shown above, that the fat and cholesterol content of the diet be reduced to the degree that will achieve the desired level of blood cholesterol.

The recommendations point to relatively minor modifications in most family diets. The changes that have been recommended require changes primarily in the fat content of the diet, which can be adjusted by the housewife with a minimum of effort. This does not suggest that the meat, dairy product, or poultry intake be reduced as such, but only to the extent that the fats of these products are partly reduced. In the case of meat or poultry, it is suggested that drippings, separable fat, and skin be discarded. In the case of dairy products, that the fat content be partly reduced; many milk products are available in these forms.

The egg is the single most controversial item in the American diet based on the proposed changes. A single egg contains approximately 260 mg. of cholesterol present in the yolk. If you eat two eggs per day, you have already exceeded the 500 mg. maximum given in the provisional recommendations. We should keep in mind that the source of eggs in the diet is both visible and invisible. Eggs are included in the preparation of many foods, hence more caution is required. In the case of dietary fats in general, it is recommended that fats high in saturated fatty acids be partly replaced by fats high in mono- and polyunsaturated fatty acids. It is further recommended that starch calories such as are present in cereals, legumes, potatoes, rice, and pastas be substituted for the decreased fat calories to maintain a desirable weight.

In the reevaluation of diets designed to contain less fat, a study of levels of fats, fatty acids, and cholesterol in average amounts of foods consumed by men, women, and children has been made. This information was gathered from surveys carried out by the USDA in past years and has served as a basis for recommending diets with reduced fat levels.

The effect of the nutritive value of average amounts of individual food items must be taken into account when adjustments are made to reduce the levels of fat in the diet.

What is the USDA Doing?

The USDA has been working in this direction for many years. Examples of progress within the USDA that relate to diet and heart disease are many. Only the most salient will be mentioned:

... Development of leaner strains of swine through genetic inbreeding.

... Incorporation of more polyunsaturated fat into dairy products and into beef carcass by feeding diets to animals containing encapsulated oils.

... Reduction of the fat content of Type A School Lunch where needed.

... Compilation and publication of new information on fats in foods to include fatty acids and cholesterol.

... Evaluation of dietary adjustments leading to decreased fat for low and moderately priced food plans and the effects on total nutritive value, acceptability, cost, and feasibility on this type of dietary change.

... Initiation of studies in man to ascertain how much meat, dairy products, and poultry can be eaten and still avoid undue elevations in blood lipids.

... Initiation of studies in man and animals to study the effect of diet on blood clotting parameters, especially those related to thrombosis.

... Development of a safe and simple method for evaluation of atherosclerosis by noninvasive techniques in man.

... Evaluation of the role that polyunsaturated fat plays in metabolic interrelationships of proteins, fats, and carbohydrates, especially through prostaglandins.

... Study of the role of simple and complex carbohydrates in the development of metabolic changes which may lead to vascular disease.

... Nutritional studies to test the interrelationships of trace elements and vitamins in the diet on fat metabolism.

These are ongoing projects having high priority and which will yield valuable nutritional information and more healthful foods.

Conclusion

This is a brief overview of the direction that this Agency is taking on the many-faceted problems of diet and heart disease. We feel that we are moving in the right direction, taking cautious steps because we are aware of the many pitfalls that we must still overcome in understanding the role of all of the nutrients and all the implications of dietary change involved in this problem. We are aware that there are several environmental factors in addition to diets as have been previously mentioned in this report, that are also involved in the development of heart disease. It is important to keep in mind that the exact roles of these nondietary factors relating to heart disease are much less understood, for the most part, than diet. They cannot yet be placed in full perspective. We view the future of nutrition and of dietary recommendations for health to the citizens of this country as being one of our primary missions. We recognize that many of the answers are still to come. In the meantime, we must make certain concerted judgments based upon information at hand for the benefit of the public.

LOW CALORIE FOODS – WHY

by
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The subject of low calorie foods is of wide interest in all of the industrialized countries of the world because, in general, the average person consumes more calories than he needs for his activity, and the excess is deposited as adipose tissue. This is especially true in the United States.

The total world production of calories is shown on the map (fig. 1). An excess of calories is produced in the temperate zones, whereas there is a deficiency in the tropical zones. This may reflect the natural forces of sociological and industrial development. It may have resulted from the need of the temperate zones to produce enough food during the growing season to carry over during the nonproductive winter months. Increased demand resulted in more food than could comfortably be consumed. People in the temperate zones consumed more than was needed, leading to the present state of over-calorie consumption.

In those areas of the world's temperate zones where excess calories are produced, there is a concomitant high consumption of calories as shown in table 1. If the average calorie consumption per area is multiplied by the population of that area and the total consumed calories added together and divided by the world population, an averaged 2,548 calories are consumed per individual per day throughout the world. This is 48 calories more per person that is considered by most nutritionists as the necessary daily average. If it is considered that the average daily consumption worldwide should be 2,500 kilogram calories, it is evident from fig. 2 that areas in the world's temperate zones consume from 100 to more than 700 calories per day in excess of their requirements. The United States ranks among the highest, consuming on the average an excess of approximately 690 calories per day.

Figure 1

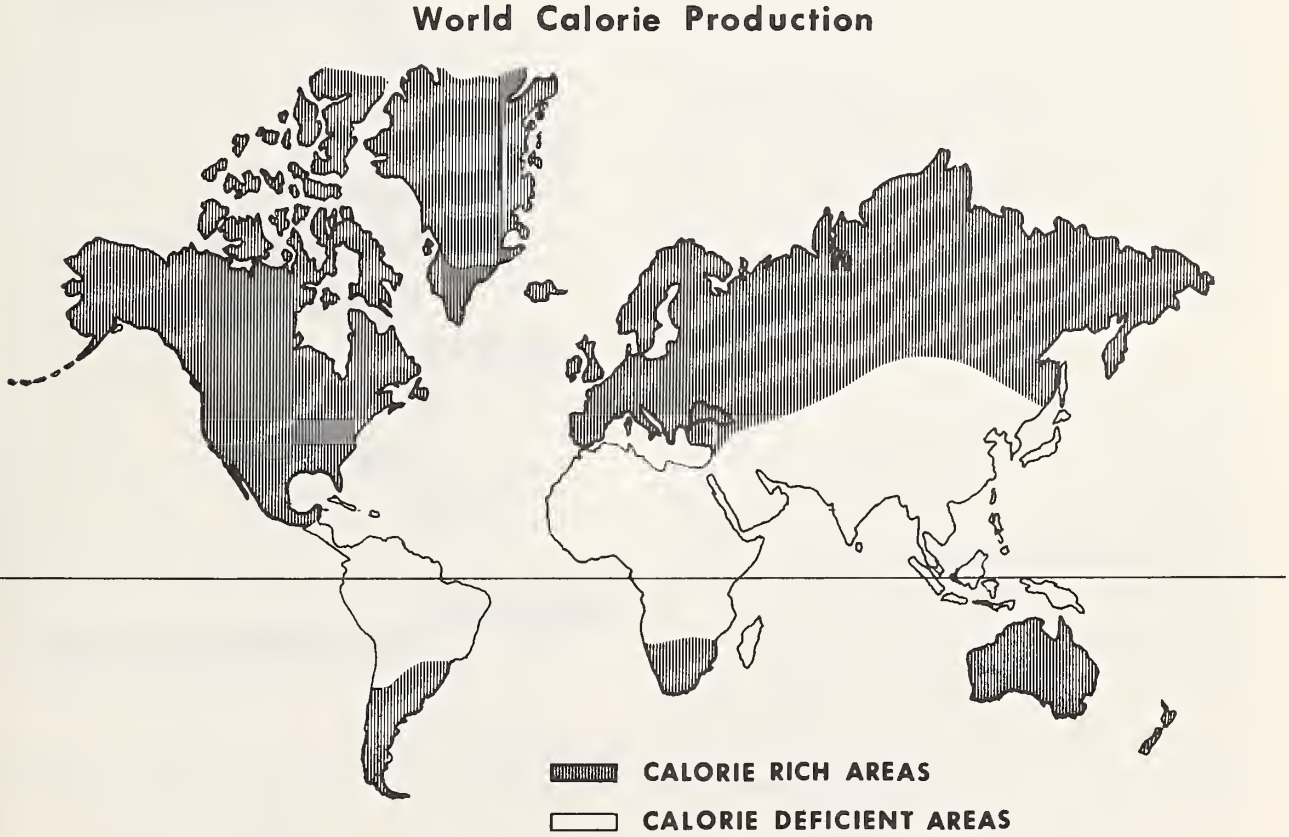

Figure 1. World calorie production

Table 1. Daily calorie consumption

Location	Calories
North America	3,170
Mexico	2,580
Central America and Carribean	2,284
Northern South America	2,205
Southern South America	2,740
Western Europe	2,981
Eastern Europe and USSR	2,934
Northern Africa	2,226
Southern Africa – (x-So. Africa)	2,434
Republic of South Africa	2,720
West Asia and Middle East	2,382
South Asia, Southeast Asia and Far East	2,120
Australia and New Zealand	3,256
Total world	2,548

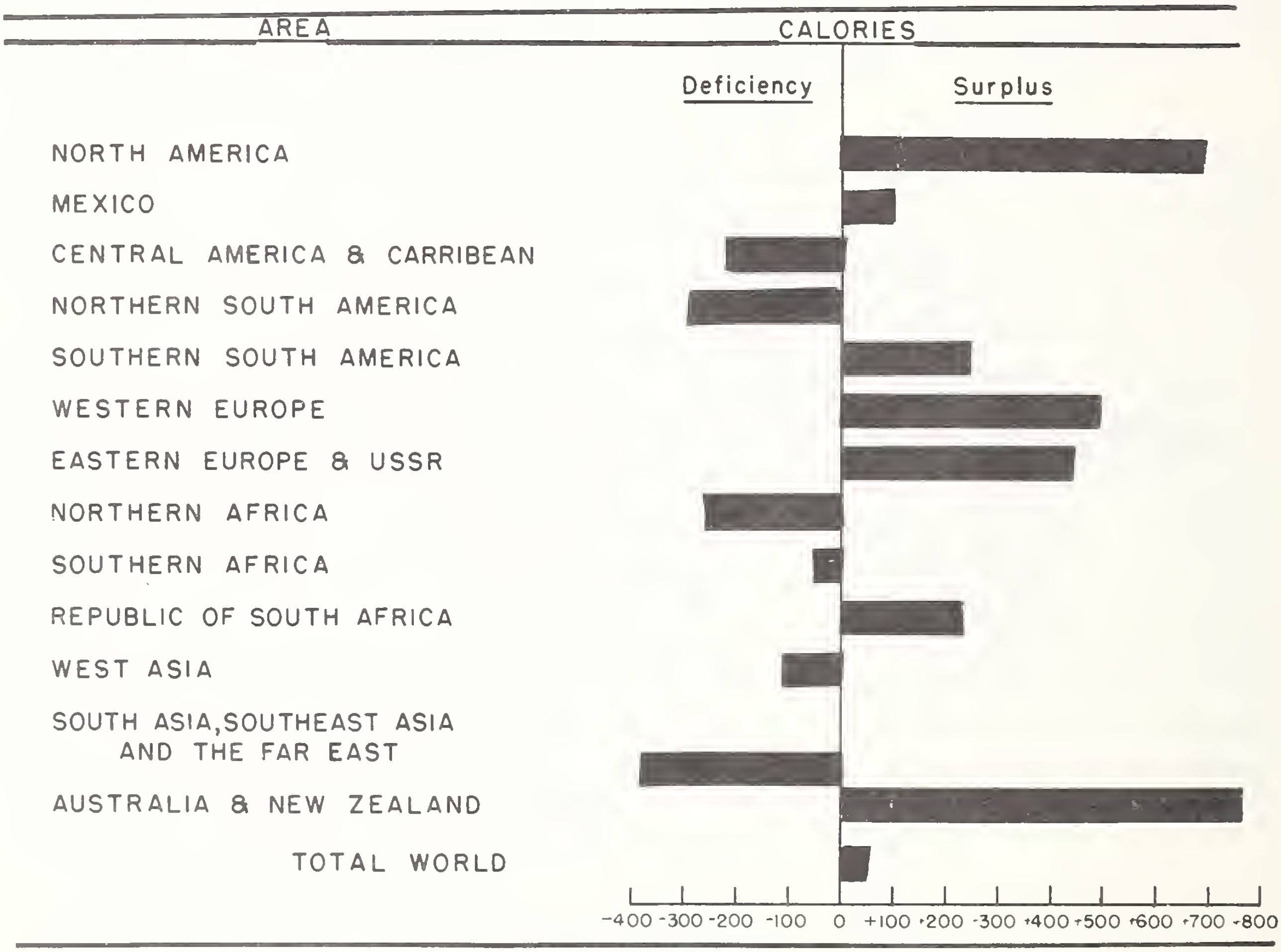

Figure 2. Comparison of calorie consumption and requirement

Although some of the United States population falls short of this intake, too many of us make up the difference. Conversely, in the tropical areas of the world there is invariably a calorie intake deficiency.

Ideally, calories would be equally distributed to satisfy demand, but this is not the case. Areas suffering from overproduction also suffer from overconsumption, and areas where there is an underproduction of calories continue to be plagued by hunger.

It is human nature to eat too many calories if they are available. Although the esthetic and cosmetic aspect provides an incentive to avoid overweight, it is increasingly evident that overweight is generally undesirable healthwise. Obesity is classified as malnutrition, and is a serious public health problem in the United States. Overweight people are more than twice as likely to have symptoms of chronic disease as those of normal weight or slightly below normal. Obese people are prone to overtiredness, shortness of breath, various aches and pains, swelling of the joints, indigestion, chronic headaches, and constipation, and these conditions are more likely to occur at a younger age. Also, overweight people are more liable to have accidents, difficulty in having families, complications of pregnancy, and stillbirths. They are four times more likely to die from major surgery than a normal weight or lean person.

There are additional adverse effects of obesity. The incidence of arthritis, diabetes, gall bladder inflammation, high blood pressure, arterosclerosis, varicose veins, coronary heart disease, cerebral stroke, and liver disease is significantly greater among stout people. In men 49 to 69 years of age, the average mortality rate is 10 to 15 percent higher for a 10 percent overweight; 20 to 25 percent for a 20 percent overweight; 40 to 45 percent for a 30 percent overweight; and 70 percent higher for a 40 percent overweight. In other words, if a man should weigh 160 pounds, but instead weighs 210 pounds, his chances of survival for the next year are about half that of the man of normal weight. Among those 20 percent overweight, 40 percent have more heart and circulatory disease, 50 percent more cerebral hemorrhage, and 75 percent more nephritis. The incidence is increased also in diabetes, gall bladder, liver diseases, and diseases of the digestive tract.

Nevertheless there is still hope for fat Americans. Evidence shows that an overweight person who reduces has practically the same mortality rate and serious disease susceptibility as persons of normal weight — provided he reduces before obesity is longstanding and no complicating disease has evidenced itself.

The problem of obesity, or its prevention, is not solved by the same treatment for all, since body chemistries differ. In general, the American diet might consist of approximately the following:

Component	Percent of Calories Required
Carbohydrates (minimum of sweets)	50
Fats (nutritionally balanced)	35
Protein (nutritionally balanced)	15
Minerals (as required)	
Vitamins (as required)	
Water	
Bulking agent	

At the 2,500 calorie per day intake level, this would mean the diet should include about 325 grams of carbohydrates, 97 grams of fats, and 94 grams of protein, together with an adequate supply of the necessary vitamins and minerals, and sufficient bulking components to carry the more essential nutrients. Obviously, also, sufficient water must be ingested to supply the normal needs of the human system.

In the case of the carbohydrate component, the greater portion should probably be starch since this form of carbohydrate is perhaps the most compatible with most total food composition product formulations, and the high ratio of starch to other carbohydrates represents the most healthful dietary composition.

One should recognize the limitations and problems associated with excessive sugar consumption. In addition to the probable satiation effect of consuming over 3/4ths of a pound of sugar per day, that much sugar would, without question, lead to increased dental caries, higher blood serum cholesterol production, diabetes enhancement, and a completely unbalanced dietary. The higher starch to sugar ratio will lend a more acceptable total carbohydrate component.

Fats, of course, represent the highest caloric component of the diet, but are unquestionably necessary in any balanced diet. Much controversy still exists over the question of optimum polyunsaturated to saturated fat ratio, but there appears to be no question of the need for an adequate supply of the essential fatty acids as well as the need for the supplemental effect of the other, nonessential fatty acids. Furthermore, evidence is growing that the cis-isomer is really the form which is biologically utilized, but there still remains the question of whether or not the trans-isomer is merely along for the ride. At any rate, fats are important and the average American diet of 2,500 calories should contain somewhere in the neighborhood of 97 grams of fats and oils.

The average daily food intake of people in the industrialized countries — particularly in the United States — includes a generous supply of nutritionally well-balanced proteins; in fact, in many cases well over 100 grams per day. Although the optimum average is 94 grams of protein, this quantity could be somewhat more or less, as is true also of carbohydrate and fat, depending on the particular requirements of the individual.

It should be stressed, however, that the protein component must be of a nutritionally balanced amino acid composition. All of the essential amino acids must be present in approximately their optimum ratio, whole egg protein usually being used as standard. In addition, it is considered important to supply the nonessential amino acids in varying amounts; but, generally at a rate of at least about two-thirds the amount of essential amino acids. In many nutritious food products, however, the ratio of essential to nonessential amino acids is considerably less than the 3 to 2 figure. With this information as a base point, we are now in a position to consider means of developing the lower calorie food products so sorely needed in our American dietary. It would be very difficult, if not virtually impossible, to require that every lower calorie food product meet all the criteria of the optimum, standard, average diet. Therefore, it's wiser to attempt to achieve the lower calorie diet in a number of different ways.

For example, one of the most useful tools for achieving lower calories is the use of nonnutritive sweeteners in the normally sugar-sweetened foods. Such use has been a Godsend to the diabetic, because the sweeteners provide added enjoyment without the risk of the ill effects of hyperglycemia. Most foods on the so-called "Dietetic Shelves" of the supermarket fall in this category, including such items as dietetic canned fruits, applesauce, jams and jellies, salad dressings, bottled soft drinks and mixes, gelatin desserts, ice cream, confections, and chewing gum. The only currently Food and Drug Approved noncaloric sweetener is saccharin and its salts. Sorbitol and mannitol, which are reported to absorb and/or metabolize more slowly and to a somewhat lesser extent than the regular, well known sweeteners, are also being used in many dietary confections and some other food products.

Little expertise, really, is required to formulate with the non-nutritive sweeteners. Processors must bear in mind, however, that when the foods are intended for use by the diabetic, it is important to be able to supply the consumer with the content of any natural sugar in the product; to be aware that the nonnutritive sweetener does not have the preservative effect of the natural, caloric sweeteners; and to balance the sweetener/flavor combination to satisfy consumer acceptability.

A second means of attaining lower calorie foods is through the use of hydrocolloids to absorb and hold more water. This added bulk of water reduces proportionally the calorie content of the food product and at the same time serves to satisfy the appetite. Some of the products in this classification are represented by the canned liquid "complete meals" of 225 to 275 calories which are sold under various brand names. The most frequently hydrocolloid is carrageenan since it tends to produce more "body" in the liquid product. But other effective hydrocolloids will serve the same purpose.

A quite unique method of attaining reduced calories in highly acceptable and sought-after food products is aeration. Whipped cream and marshmallow are perhaps the two classic examples of the use of aeration to develop an unique and highly acceptable texture with the resultant considerably reduced calories per volume of food served. More recently, this method of attaining lower calorie products has resulted in extensive use of the aerosol dispenser, not only for whipped creamlike products but also for cheese spreads, omelette compositions, sauce mixes, and a host of similar products readily adaptable to this technique. The great multitude of puffed snack items represents still another very useful means of attaining bulk through aeration.

Non-caloric bulking agents are also beginning to assume a measured degree of importance in the development of lower calorie foods. For example, purified cellulose is now being used as a filler in amounts significant enough in food composition to result in up to 25 percent to 30 percent reduction in caloric content. Natural physiological characteristics, such as constipation and possible absorption of essential nutrients, limit the quantity of such fillers in food product formulations. The fact that defatted soybean flour contains a fairly high proportion of nondigestible polysaccharides makes it a useful ingredient in many food compositions where a lower calorie content is sought. The use of defatted soy flours in crackers, cookies, and other bakery products are excellent examples of achieving lower calorie foods and supplying a good source of high quality protein.

Since fat represents the highest caloric component of foods — 9 calories per gram as compared with 4 for either carbohydrates or proteins — it appears quite obvious that removing fat from natural food products is one means of reducing their caloric content. The separation of cream from milk is, no doubt, the oldest classical example of this method of calorie reduction. Since butter fat represents almost one-half the total calorie content of whole milk, the skimming or cream separation process represents one of the most efficient means of production of a lower calorie food.

More recently, fat extraction from a natural product has been exemplified by the process developed here at the Southern Marketing and Nutrition Research Division. Your removal of a large percentage of the oil from peanuts to yield a low fat and thereby a lower calorie peanut has produced a delightful snack product which is today enjoying substantial sales and consumer acceptance.

Reformulation of well known fabricated foods relatively high in fat content, in such a way that the fat content is measurably diminished, is another means of accomplishing a lower calorie food product. A classical example of this type of calorie reduction resulted when the dairy industry developed the so-called water ices and ice milks as a replacement for ice cream to satisfy the calorie watchers. Many formulated food products lend themselves to similar reformulation with a consequent calorie reduction and possible even greater acceptance by the consuming public.

Similarly, newly formulated, engineered, or technically designed food products can take advantage of the combined knowledge of the nutritionist, the dietitian, the food scientist and technologist, the food engineer, and the marketing specialist. Together, these specialists can develop more stable, more nutritious, more acceptable, and better food products to supply the consumer those attributes most sought after in lower calorie foods.

It is important to remember that the term, "low calorie food," is relative. A low calorie diet may simply consist of less food, or it may constitute a multitude of products lower in calorie content than their well-known counterparts. It is important to satisfy total nutrition. This is possible at almost any daily calorie intake level provided that the calorie intake supplies the energy output needed by the individual.

With today's knowledge there is little excuse for the continuing blight of malnourished obesity, except for the innate traits of human beings to ignore common sense practice of moderation.

FOOD AND NUTRITION SERVICE'S ROLE IN DEVELOPING NEW FOODS FOR USDA CHILD FEEDING PROGRAMS

by
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ABSTRACT

The food processing industry has been cooperating with the Department of Agriculture in tailoring new foods for Government food programs. One group of new foods is for our Child Nutrition Programs. At present about 25 million children are eating lunches in 82,000 schools. This number could be expanded if convenient, nutritious, palatable, low cost foods were available for special needs. Textured vegetable proteins lower meal costs, as does the high protein macaroni. Cup-cans allow schools with no facilities to serve a hot meal — even though kitchens and cafeterias are not available. Formulated bakery products expand the use of the Breakfast Program to schools without facilities.

Keywords: Engineered foods, fabricated foods, formulated foods, fortification, enrichment, child nutrition programs.

INTRODUCTION

Our foods are changing, just as we are changing. New and improved techniques continue to alter our food production methods. Mechanization, fertilization, irrigation, hybrids, and pest control — all contributed to transforming farming more in the last 60 years than in its entire history — thus helping make it possible for the United States to have the most abundant food supply in the world.

Changes of similar impact have also come to food marketing. New concepts, processes, packages, handling methods have resulted in convenience foods that have changed both home and institutional feeding operations.

We now move one step further and have entered into another phase of change — fabricated or formulated foods. These are food ingredients or food components that have been put together in a new form or a new composition — usually as an answer to a specific need or problem. They may also be familiar foods redesigned to meet our needs.

Convenience, simulated, and synthetic are names we use to describe various new foods. The Food and Nutrition Service (FNS) has not and shall not accept totally synthetic foods for use in its Child Nutrition Programs. These are foods developed from nonnatural sources and replace natural foods. Powdered fruit-type drinks from nonfruit sources is an example.

All formulated foods accepted for use in our FNS programs come mainly from natural foods, such as flour, fats, egg, and nonfat dry milk. In this way the end product will contain the trace elements found in these natural ingredients. In addition, the foods are not served alone, but are part of a total meal.

I am here today to discuss the Department's interest in formulated foods, and how they may be used in our various feeding programs. I am not here because we know all there is to know about formulated foods, but rather to share the knowledge and experience we have gained, and to get your suggestions, ideas, comments, and criticism on our approach to the use of formulated foods in the Child Feeding Programs.

A question often asked is "What is the Department's interest in formulated foods?" To help you understand, I will try to give you some background information on the subject.

The background. — In 1969, the Panel on New Foods of the White House Conference on Food, Nutrition, and Health recommended:

> "implementation of an immediate food fortification program to relieve malnutrition."

That same year, President Nixon stated his intent to eliminate hunger and malnutrition in the United States.

Even before this, in 1968, the Food and Nutrition Board of the National Research Council, and also the Council on Foods and Nutrition of the American Medical Association recommended:

> "that efforts be continued to develop new and improved foods that will provide superior nutritional quality, greater variety, and acceptability — and when important, be economically advantageous."

So here was the challenge:

Feed hungry children and feed them now!

Where?

In remote areas! In old schools without kitchen facilities! In places where children must travel long distances to school without breakfast!

How? That was the question.

Food technology was one answer to improve the nutritional quality of foods served in our USDA Programs.

The Food and Nutrition Service called on the food industry to make:

> "nutritious foods widely available in popular form."

and industry responded.

Even though a properly balanced diet can come from a variety of conventional foods, we were aware that many families, schools, and institutions did not have adequate facilities for preparing and serving conventional foods. So, for this reason, we are interested in promoting foods and food concepts that can improve the nutritional quality of our programs or help our programs to reach eligible individuals and groups who are not now being reached because of a lack of facilities.

Another question frequently asked is how we accept new products? What is our procedure?

FOOD EVALUATION COMMITTEE

An FNS Food Evaluation Committee was formed about seven months ago. Its membership is composed of nutritionists, home economists, food technologists, and economists within the Nutrition and Technical Services Staff, plus representatives from FNS Child Nutrition and Technical Services Staff, plus representatives from FNS Child Nutrition and Food Distribution Divisions.

One of the Committee's first endeavors was to prepare a Position Statement on Formulated Foods and the Committee's operations and procedures for evaluating new foods.

The Food Evaluation Committee has responsibility to:

1. Define food components within established meal patterns for which each specific new food can serve as an alternate.
2. Examine and evaluate some new foods and concept in their developmental stages and make appropriate recommendations.
3. Determine the contributions and limitations for each new food and new food service concept as related to the Department's present food programs.
4. Determine necessary modifications and recommend changes to present food program regulations to enable inclusion of new food items or new food service concepts.
5. Evaluate acceptability and effectiveness of accepted new foods and new food service concepts used in FNS programs.
6. Recommend changes in existing food product specifications to comply with recent advances in nutritional knowledge and food processing techniques.
7. Set standards and criteria for implementing procedures to monitor compliance of specifications and requirements for all accepted new foods and new food service concepts.
8. Determine the necessary educational materials required to understand the nutritional implications of the new foods and new food service concepts by the Department.

Nutrition education is an area that needs our immediate attention. Members of the Committee have discussed the preparation of flyers on products that have been accepted to date. These could be distributed to schools using the products. The flyers would explain the nutritional value of formulated foods and how the values differ from the traditional food. They would also give reasons for the development of formulated items and how they apply to our programs.

We believe that manufacturers of these new products also share the responsibility to provide educational programs to inform the public of nutritional differences.

Our criteria for evaluating new foods to be considered for our programs are: improved nutrition; acceptability to the consumer; convenience in terms of labor time required for preparation, service, clean up and improved product stability; and cost in terms of the effect on the total cost of the meal. Before a product is accepted, we ask the food processing company involved to provide the results of an independent laboratory analysis for nutrient content and biological value.

The Committee may accept only those foods and food service concepts ultimately intended for the general population through commercial markets and not those products solely available for Government programs.

This Food Evaluation Committee recommends new foods to our Administrator. If further evaluation is not required, the food is placed on an appropriate list of accepted foods which is made available to Regional Offices, State Agencies, and industry.

AUTHORITY FOR NEW FOODS

At this point I digress for a moment and briefly discuss our authority for accepting new foods into our programs. In Child Nutrition Programs, for example, regulations define the meal requirements for three programs: Breakfast Program, National School Lunch Program, and the Special Food Service Program for Children. The Type A lunch of the National School Lunch Program is to include 2 ounces of cooked meat or meat alternate, 3/4 cup serving of 2 or more fruits and/or vegetables, 1 serving of bread, 1 teaspoon of butter, and 1/2 pint of fluid whole milk. Any new food that conforms to existing meal regulations may be accepted and used in schools without testing or evaluation. Some new foods do not fit into present regulations. For example, if a plant protein product is to be considered as a meat alternate, it requires testing and may even require a new Notice to define its use and composition.

NEW PRODUCTS AND CONCEPTS

During the past year, we have written five product specifications and have several more in various stages of preparation. These include:

Textured Vegetable Protein (FNS Notice 219)
Fortified Macaroni (FNS Notice 218)
Cup-Cans (FNS Notice 270)
Fortified Baked Products (FNS Notice 180)
Formulated Grain-Fruit Products (Instruction 783-5)

FNS Notice 180 above, has been replaced by the Formulated Grain-Fruit Product Instruction 783-5. Proposed product specifications are granular protein concentrated, protein isolates, bread products, high protein, puddings, and dairy-fruit combinations.

We now discuss some individual products, such as the cup-cans, textured vegetable proteins, fortified macaroni, and formulated bakery products. These Notices will serve as prototypes for our present evaluation and acceptance processes.

1. Cup-Cans. — Last summer (1971), the Philadelphia Archdiocese asked special permission to use canned vending machine foods in their private schools. Located in crowded downtown areas, the schools did not have cafeterias or kitchens. Yet the religious authorities wanted the schools to be able to qualify for USDA reimbursement under our Type A Lunch Program. They also wanted a low cost hot lunch for their children. Regular vending machine canned items were tried first. The products were heated in the usual vending machine. The enameled-lined cans were opened with a can opener.

The cup-can entree is now in Phase 2 of its development, and starting into Phase 3. Improvements are being added continually. Here are some characteristics of the systems and concept.

a. Children eat at their desks, directly from the can. Plastic spoons, paper plates, and napkins eliminate dishwashing and pots and pans.
b. Easy-open lid.
c. Cans are heated to 140° to 150° F. Heating starts the night before. The heating machines hold 80 to 336 cans, use 110 volt current, and cost $125 to $400.
d. Products are tailored for children's rather than adult's tastes, i.e., the chili con carne is milder than an adult version.
e. New products are fabricated so that each can contains a full 2 ounces of cooked meat or meat alternate and 1/4 cup of vegetables.
f. The label states the can's contents relative to our Type A lunch requirements.

I mentioned cup-cans were now in Phase 2 development. The cup-can innovations expected and being tested for Phase 3 include:

a. New can design. A squat can now being tested is more easily heated and more nearly resembles a bowl.
b. A new type easy-open lid that will eliminate sharp edges thus avoiding injury if children lick the lid.
c. A holder for the can.
d. New label and new inspection procedure.
e. Use of high protein macaroni as a partial meat alternate.
f. Use of textured vegetable protein as a partial meat replacement.
g. Use of dry beans as both a meat alternate and as a vegetable.
h. Development of supplemental foods to go with the cup-can. Example: a butter-biscuit that meets the bread and butter requirements of Type A lunch.

2. High protein macaroni. — There is no FDA Standard of Identity for high protein macaroni, although the product was given a temporary marketing permit over a year ago. High protein macaroni that qualifies for use in our programs is different from all others in that we do not specify ingredients. We do ask that it meet certain nutritional specifications as follows: PER 95 percent of casein; protein, 20 to 25 percent; iron, 13.0 to 16.5 mg./lb.; thiamin, 4 to 5 mg./lb.; riboflavin, 1.7 to 2.2 mg./lb.; and niacin, 27 to 34 mg./lb. As a meat alternate it may replace up to 50 percent of the meat when served with meat.

   General Foods Corporation, the first firm to place an acceptable USDA macaroni product on the market, has had several formulations. Their first formulation was 60 percent corn, 30 percent soy flour, and 10 percent wheat. Their present formulation is about 38 percent corn, 30 percent soy flour, and 30 percent wheat. The Prince Macaroni Company's formulation is 78 percent wheat, 8 percent soy flour, 5 percent wheat germ, 5 percent wheat gluten, and 4 percent other ingredients. It is noted that lysine is added to their product to boost the biological value of the protein.

   In school tests the product appears quite acceptable in tomato products but is less acceptable in cheese mixtures. It is now being tested in Lubbock, Corpus Christi, Brownsville, Dallas, Fort Worth, and Houston, Tex. Present tests are now being undertaken in New Jersey, Delaware, and Maine.

   Although the future of fortified macaroni has been uncertain to date, a Standard of Identity from FDA would help establish its future. In USDA, priority will be given to promoting the use of this product in child nutrition programs. The new macaroni will give schools still another choice of high protein food at lower cost.

3. Textured vegetable protein. — This product is approved as a meat alternate when no more than 30 percent hydrated textured vegetable proteins is mixed with 70 percent red meat, poultry, or fish. Probably no other new formulated food has achieved wider publicity. Opponents call it an "ersatz" meat, substitute, or filler. Others see it as the protein of the future. It is the first plant protein to come close to meat in nutritional value. One reason for the high nutritional value of the product is that we require mineral and vitamin fortification.

   Two measures of protein are its quantity and quality. In quantity, hydrated textured vegetable protein has 20 percent protein. I believe that most of our ground meat now being used in school lunches is 16 to 18 percent protein. In terms of protein quality, textured vegetable protein has a PER of at least 1.8, with much of it in the PER range of 2.0 to 2.2. In comparison, beef has a PER of 2.6 to 2.8. When the textured vegetable protein product is mixed with meat the PER of the mixture is probably about the same as meat served alone.

   No one knows the volume of textured vegetable protein now sold in schools, but certainly its use is increasing rapidly. I wish to stress that textured vegetable protein is a meat supplement rather than a meat substitute.

   Several months ago we released our first list of companies and brands of textured vegetable protein products that met our FNS Notice 219 specifications. Our second list came out in March 1972. The purpose of this list is to help schools and State agencies select those plant proteins that are acceptable under FNS Notice 219.

   FNS Notice 219 is now being revised, and a new Notice on Granular Protein Concentrates will help define these two plant proteins for future use. Before we leave the subject of plant proteins, I would like to speak on the question of whether these plant protein products have the future potential of replacing meats in our economy. My thoughts do not necessarily reflect the thinking of USDA.

a. In the years ahead the U.S. livestock industry will not be able to supply the proportion of proteins that it now provides. Thus, as a Nation we will be considering various ways of adding to our protein supply. Some immediate choices will be to: import meat from other countries; use certain marine proteins; use proteins derived from fungi, yeasts, and other microbiological sources; use plant proteins. I believe adding U.S. produced plant proteins to supplement U.S. produced animal proteins is a wise move from a national point of view.

b. Plant proteins may actually be able to help rather than hurt the meat industry in the sense that, when added to lower cost meats as ground meat, they can extend these meats and make them more useful. They retain the fats and moisture during cooking thus making a juicier and more flavorful product.

4. Formulated grain-fruit products. — FNS is interested in making our school breakfast program available to more children. Our original specifications (FNS Notice 180) came out in the fall of 1970 and attempted to accomplish this objective. But for the first year we had only one product, Astrofood, from Continental Bakery. As one might expect, monotony became a problem.

Last month (March 1972) we released Instruction 783-5. As a result of changes, we anticipate a wide variety of products submitted for acceptance.

What is the purpose of this new Instruction and what needs will it fill? Our Breakfast Program requires three food components: 1/2 pint fluid whole milk, juice/fruit, and bread/cereal. The new foods submitted for acceptance will both meet the juice/fruit and bread/cereal requirements, thus combining two foods into one and facilitating serving food in schools without kitchen and serving facilities. The resulting grain-fruit products will be high in nutrients and many of them will contain actual fruits or juices.

Fruit pies, fruit tarts, fruit cookies, fig bars, doughnuts, peanut butter cookies, cup cakes, wafers, cookies, and crackers should be developed. These foods will be used not only in breakfast, but will find a place in the Special Food Service Program for Children as a supplemental (or snack) food as well. Our summer feeding program will most likely use many of them as they become accepted.

SOME FUTURE FOODS

In a meeting such as this, we should at least mention some future food prospects. We have considered a new peanut butter that has a PER of over 2.0 (regular peanut butter has a PER 1.2 to 1.3). If accepted for use, the new peanut butter will need a new Standard of Identity and probably a new name. In child nutrition programs it will provide a higher quality protein (when peanut butter is used as a meat alternate). In family feeding programs it will supply more protein to those needy families using it. The new peanut butter is less sticky than the regular one.

Several companies have developed high protein puddings, and new product specifications may be written to allow their use in our programs. Dairy firms have developed fruit/milk products and milkshake mixes that we intend to evaluate in the near future.

Breakfast cereals have been severely criticized recently. We take this opportunity to invite cereal manufacturers to work with us in developing or testing nutritionally upgraded cereal products. Some may be already developed awaiting a market.

I have limited my remarks to new foods and concepts related to USDA's child nutrition programs. Other foods in Government Distribution Programs are of equal importance and deserve equal time, but I will not discuss them at this time. The foods of the future are changing rapidly.

SELENIUM AS AN ESSENTIAL ELEMENT

by
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The discovery by Schwarz and Foltz (10) of the nutritional importance of selenium has led to an increasing amount of research on its mode of action in preventing nutritional diseases as well as on its deleterious effects. Today, I would like to talk about some of the work I did at the University of Wisconsin in conjunction with W. G. Hoekstra, A. L. Pope, and H. E. Ganther which suggests a biochemical mode of action for selenium. In addition, I would like to discuss the implications this work might have on animal and human health.

The need to define a metabolic role for selenium arises not only from its nutritional importance, but also from a need to explain its interactions with vitamin E and the sulfur amino acids. Tappel (13) thought that all three of these nutrients can act as antioxidants. His view, however, has not been uniformly accepted by the scientific community. Our work suggests a biochemical mode of action for selenium and offers some new insights into the interactions of selenium, vitamin E, and the sulfur amino acids. The experiments described below led us to conclude that selenium participates in an enzymatic reaction. We have demonstrated that dietary selenium protects against hemolysis and oxidative damage to red blood cells in vitro provided that glucose is included in the incubation medium (6,8,9). Since hemolysis can also be prevented by vitamin E, this work provided an in vitro assay system which demonstrated overlapping functions of vitamin E and selenium. To the contrary, oxidative damage to hemoglobin was not prevented by vitamin E, thus providing an in vitro assay in which the functions of vitamin E and selenium are independent of each other. Interestingly, the well-known effect of glucose in preventing hemolysis and oxidative damage is known to depend on the ability of glucose to maintain the level of reduced glutathione (GSH). This effect is due to the combined actions of the enzymes, glucose-6-phosphate dehydrogenase, and glutathione reductase[2]. The effect of GSH in preventing hemolysis has been attributed to its participation in the enzymatic reaction catalyzed by GSH peroxidase (2 GSH + H_2O_2 - $2H_2O$ + GSSG) (2). Thus, glucose through its ability to maintain GSH plays an important role in destroying peroxides which are known to cause hemolysis and other types of oxidative damage. More importantly, the interaction of dietary selenium and glucose in preventing oxidative damage and hemolysis, implies a relationship between peroxide destruction and dietary selenium. We subsequently demonstrated that GSH peroxidase activity is dependent on dietary selenium and have preliminary evidence to suggest that selenium is a constituent of GSH peroxidase (7).

The observation that dietary selenium is essential for GSH peroxidase activity allows us a way to account for the close interaction of selenium with sulfur amino acids (as a precursor of cysteine which is an integral part of GSH-y-glutamyl-cysteinyl-glycine) and vitamin E. If vitamin E acts as an antioxidant (that is, prevents peroxide formation) and both selenium and the sulfur amino acids (as a precursor of a part of GSH) exert their influence on peroxide destruction, one can readily envision an overlap of function of these three nutrients in preventing the same diseases. The in vitro assays described in which selenium does and vitamin E does not prevent oxidative damage allow us to discriminate between the effects of selenium and vitamin E. This provides a precedent for explaining the diseases where the nutritional effects of vitamin E and selenium do not overlap.

Selenium in Animal Health

In addition to the effect of selenium in preventing nutritional diseases in laboratory animals, it has been shown to prevent exudative diathesis in chicks, myopathies of the gizzard and heart in turkeys, white muscle disease in lambs and calves, hepatosis dietetica in pigs, and to improve reproduction in cattle and sheep (14). It has been estimated that selenium supplementation would add $10,000,000 income to the livestock producers in the Northwest (U. S. A.) alone[4]. A survey has indicated that selenium deficiency is a problem in at least half of the States, and selenium deficiency is known to be a problem in many of the major livestock producing areas of the world (4).

Although the effect of selenium in preventing a number of diseases is well known, its use has been forbidden because of its listing as a carcinogen. In view of the recent work implicating selenium as a possible agent against human cancer (12), the question of the carcinogenicity of selenium needs critical re-examination. It is to be emphasized that the evidence on this matter is by no means final. Another concern is the toxicity of this element. Although few elements are as potent at such low levels, the range between beneficial levels and toxic levels of this element is comparable with other trace minerals.

Usually the first sign of selenium deficiency is illness or death of the animal. This points to a definite need for a good specific clinical indicator of selenium deficiency where it is a problem, or where it may be confused with other diseases.

Although Kubota and others' (3) data show selenium deficient areas it is clear that selenium deficiency is not always directly related to selenium levels (1), but can be dependent on availability or influenced by antagonistic factors. A clinical assay for selenium deficiency would provide a means for identifying the areas where selenium deficiency would provide a means for identifying the areas where selenium deficiency is a borderline problem or where availability or antagonistic factors may lead to selenium deficiency.

Although selenite and selenate have been used most frequently to prevent selenium deficiency, an organic form of selenium is three to four times more active than selenite (11). This organic form of selenium, which has not been identified, in addition to being more potent than selenite selenium would also be safer, at least in proportion to the lower dosage of selenium required.

We believe that if the relationship of selenium to GSH peroxidase continues to be valid an assay of this enzyme will serve as a useful means for defining the required level of selenium, for identifying and detecting selenium deficiency, and for clarifying the possible role of selenium as a cancer preventing agent. It is also our hope that this work will serve as a useful tool for identifying the active form of selenium.

Selenium in Human Nutrition

In spite of the large amount of evidence strongly indicating that selenium is an essential element for all species, there has been little data to suggest that selenium deficiency occurs in humans. Presently feed manufacturers and animal producers are very concerned about selenium deficiency. Mindful that many parallels in animal and human diseases occur we may expect that in a few years we could find selenium deficiency in humans. If humans suffer from selenium deficiencies what are the signs and how could it be clinically evaluated? If the relationship between selenium and GSH peroxidase holds for humans, a simple assay of this enzyme in blood could serve as a clinical indicator of selenium deficiency. Since it is known that a genetic deficiency of GSH peroxidase in humans leads to an increased susceptibility to hemolytic anemia (5), one might explore the unexplained hemolytic anemias for selenium deficiency.

Summary

Evidence has been presented supporting a specific biochemical role of selenium. Rats deficient in selenium have been shown to have reduced levels of glutathione peroxidase activity. This observation may help solve many of the problems related to selenium deficiency in animal health and could be useful in detecting selenium deficiency in humans.

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MICROBIAL SURVIVAL DURING CLEANING OF TEXTILES

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The U. S. Department of Agriculture has been concerned with the hygienic aspects of textiles as early as 1930. At that time, a bibliography relating to clothing and health was compiled. Papers published since 1875 in French, German, and English were surveyed. Of the some 1000 old references, only 38 dealt with clothing as possible fomites and these were mostly pseudo-technical in nature. The need was thus identified for scientific information upon which recommendations could be based for the proper hygienic care of textiles. Specific knowledge needed includes: factors influencing the contamination of materials; factors influencing survival and infectivity; and conditions under which bacteria and viruses are released. The purpose of this paper is to briefly review the program of the Department in this area.

Because quaternary ammonium compounds were known to possess antimicrobial activity, research was undertaken on the bacteriostatic, germicidal, and sanitizing action of these materials. A series of five typical quaternaries were selected and studied to determine their ability to impart to cotton a residual bacteriostatic action to inhibit growth of Proteus mirabilis, which commonly occurs in urine and feces. The practical objective was to prevent the formation of ammonia from urine by the urease activity of this organism and thus prevent "ammonia dermatitis," or diaper rash. All quaternaries studied were effective as a residual chemical in fabric for this purpose. For best results during simulated laundering, concentration, temperature of application, and exposure time for each quaternary varied.

The five quaternaries, at appropriate concentrations, were also found to reduce Escherichia coli counts by 99.9 percent in rinse waters of experimentally contaminated fabrics when laundered in a laboratory launder-ometer. A laundering temperature of 113° F. was more effective than 68°. When the fabric was tested directly, however, only four of the quaternaries sanitized the cotton fabric when laundered at the higher temperature; three at the lower temperature, and one was ineffective under any conditions of temperature or concentration.

In an extension of the work, using E. coli, lower concentrations of a selected quaternary were needed for sanitizing wool fabric than were needed for cotton; the reverse was true for chlorine. In most instances, both cotton and wool inoculated with Staphyloccocus aureus required a higher concentration of both of these germicides than was needed for E. coli. In general, it was found that bacteriostatic properties were imparted to cotton more readily than to wool. Although wool removed from solution more of the quaternary than cotton, the bacteriostatic property was inferior.

Fabric weave also influenced the amount of quaternary absorbed, because loosely woven cotton fabric absorbed more than tightly woven fabric. In addition, fiber type and relative humidity appear to affect survival time of different bacteria. In reference to fiber types, Staphylococcus aureus can survive on wool blankets for 18 weeks, on cotton for 12 weeks, and on acetate tricot for 7 weeks. It was later found that this order of persistence was generally true for a number of bacteria and viruses.

Up to this time, germicidal effectiveness of textiles usually was evaluated by introducing the whole treated fabric, or its extract, into an appropriate culture medium, followed by incubation. A new method was introduced which involved maceration of the treated fabric in a Waring Blendor and inoculating this extract into a culture medium.

This procedure was more effective in dislodging and recovering the bacteria, thus indicating that the morphology of fibers plays a role in the persistence of bacteria on textiles. Using this technique, it was demonstrated more forcibly in laboratory-type experiments that bacteria could survive on textiles for sufficient periods of time to be of epidemiological importance.

On a more practical level, a series of actual in-use studies was conducted on clothing from nine families, to which small swatches of fabric were attached. Home-type laundering procedures were used to obtain some indication of real-life situations. In this series of experiments, over 1500 colonies of bacteria were obtained by plating samples of macerated fabric swatches and of wash and rinse water in nutrient agar. Examination of 400 of these resulted in identification of some 30 species of bacteria belonging to 13 genera. The wash water contained from 2.5 to 10 million bacteria per ounce of water, depending on the temperature setting, and the rinse water from 960,000 to 1.2 million. Further, the bacteria were transferable to sterile fabrics during the wash. However, quaternary, phenolic, and chlorine disinfectants significantly reduced the number of bacteria remaining on textiles in the home-laundering process, frequently to a safe level.

Based on the above work, Home and Garden Bulletin No. 97 "Sanitation in Home Laundering" was issued by the U. S. Department of Agriculture. This bulletin has proved very popular with housewives, home economists, and extension workers. It sets forth practical guidelines for sanitizing textiles during home laundering, should there be a need, such as an outbreak of infection in the household. The bulletin has been updated from time to time since it was originally issued in 1964. Because of the use and popularity of the bulletin, continuing research was undertaken to develop the necessary information to maintain the guidelines current and practical. At the same time, the further objective of obtaining a broader understanding of the dissemination of microorganisms by textile fomites was sought. With this in mind, a systematic study was undertaken to determine the persistence of microorganism survival, using Salmonella typhimurium, Staphylococcus aureus, faccinia and polio virus on fabrics. These were selected because they represent a broad spectrum of microorganisms important from a public health standpoint and they differ widely in their properties. Salmonellae are gram-negative bacteria, and staphyloccoci, gram-positive. Vaccinia virus is lipophylic and a relatively large virus. The polio virus is hydrophylic and small. In addition, each of these react differently on exposure to chemical disinfectants.

The initial phase of the work involved quantitative measurements of survival of the organisms under varied environmental conditions. Artificially contaminated cotton and wool textiles of different fabric construction and different chemical treatment were maintained at 35 and 78 percent relative humidities. Contamination exposure systems involved textile dust, aerosol, and direct contact.

In all cases, survival time was greatest on wool, followed by untreated cotton, with treated cottons, exhibiting the shortest persistence time. Admittedly, fabric weave influences persistence, and the difference between wool gabardine and wool blanketing can be partly explained on this basis; the loose weave of the wool blanketing allowing greater penetration and holding power for the organisms. Where untreated and resin-treated cotton is concerned, it may be possible that the triazone resin per se had some influence on the shorter survival time. However, these resins are known to release formaldehyde, especially when a magnesium chloride catalyst is used, as in this treatment. It is highly probable that the evolution of formaldehyde was the dominant reason for the markedly reduced persistance.

In respect to relative humidity, the polio virus loses viability rapidly at the low humidity, while the vaccinia virus is stable. The vaccinia showed a low titer at 78 percent relative humidity. Salmonellae and staphylococci also lose viability at the high humidity.

As to the method of contamination, salmonellae persisted longer when applied by direct contact or in dust; staphylococci longer in aerosol and dust; polio longer by direct contact and aerosol; and vaccinia showed no difference between the three methods of application.

Other experiments were designed to determine the effect of home laundering on fabrics artificially contaminated with polio virus. It was demonstrated that polio virus could be transferred during regular laundering, although the laundering process considerably decreased virus titer. It was suspected that detergents and the high dilution in the wash and rinse cycle aided virus removal during home laundering. It was shown again that a hot-water wash was significantly more effective than the warm- or cold-water wash setting on the washing machine. This observation suggests that cold and warm water only physically remove the virus, while hot water additionally inactivates the virus. The detergents, in lowering surface tension, may have assisted somewhat in this physical removal.

Research is currently underway at WARF Institute Inc., supervised by USDA's Textiles and Clothing Laboratory, on the sanitizing effect of home laundering of a variety of modern textiles. Composite inoculations of four microorganisms were used. Laundering parameters investigated are water temperature, detergents, and disinfectants. At this stage of the research, the following conclusions can be drawn:

1 —Best overall reduction of microorganisms occurred with 100 percent cotton finished with a durable-press resin, dimethylol methyl carbamate, and zinc nitrate hexahydrate catalyst.

2 —The 100 percent cotton fabric treated with a tetrakis (hydroxymethyl) phosphonium hydroxide-ammonia flame retardant finish consistently showed the poorest reduction.

3 —The best overall treatment in the hot water phase (120° F.) was the double level of chlorine and the anionic detergent. In the warm water phase (95°), the double level of chlorine bleach with nonionic detergent was the superior treatment.

For many years, it was believed that drycleaning was a germicidal process and could be used as a means of sterilizing fabrics. Because no quantitative data were available on the extent of microbial contamination, survival, and transfer during drycleaning, limited experiments were undertaken by the Department on such processes in a commercial drycleaning plant using naturally soiled textiles. The data indicated that, under ideal conditions, the bactericidal action of the solvent, mechanical removal of cells, heat of drying, and steam finishing and pressing, combine to kill or remove the majority of bacteria present. Steam finishing and pressing appear to kill more bacteria than any other stage of the process. A few strains of bacteria that redeposited on sterile control fabrics in the study belonged to genera in which some species are pathogenic, such as Staphylococcus or Corynebacterium.

In an extension of this work, a project has recently been completed at the Southern Research Institute, under the sponsorship of the U. S. Department of Agriculture, to obtain quantitative data on the ciricidal effectiveness of standard laboratory and commercial-type, coinoperated drycleaning processes. The viruses, fabrics, and drycleaning solvent systems selected for these experiments were chosen with care, to provide the most useful information for direct application to the actual public health problem. Suspensions of polio virus, rhino virus, herpes virus, and vaccinia virus were applied to a variety of modern fabric substrates. The more significant results may be summarized as follows:

1 —The commercial, automatic machine drycleaning process was found to be superior to the standard laboratory drycleaning process in eliminating polio virus from contaminated fabrics, but not in eliminating vaccinia or herpes viruses from contaminated fabrics.

2 —Large reductions in virus titers were observed when the viruses were placed on fabrics treated with a durable-press finish, reflecting the possible inherent viricidal properties of such fabric finishes.

3 —The addition of Staticol, a commercially available drycleaning detergent, to the perchloroethylene resulted in a marked enhancement of the reduction of vaccinia virus and herpes virus titers on all fabrics during the standard laboratory drycleaning process, whereas the reduction of polio virus titers during drycleaning was generally not affected by the addition of detergent.

4 —The binding of viruses to fabrics apparently protects the agents from inactivation by fluorocarbon or chlorinated hydrocarbon drycleaning solvents.

5 —The heating of commercial drycleaning solvent containing free polio virus, rhino virus, vaccinia virus, or herpes virus for 1 hour, at 140° F., resulted in complete loss of infectivity.

6 —Infectious virus could rarely be recovered from, or detected in, samples of drycleaning solvent taken from the base storage tank of a Norge commercial drycleaner immediately following the processing of virus-contaminated fabrics.

7 —Greater reduction in virus titers was found on predrycleaned fabrics than on undrycleaned fabrics. This may be due to either (a) the viricidal properties of the residual perchloroethylene on the fabric, (b) the facilitation of physical inactivation by removal of lipoprotein impurities which might have ordinarily protected the virus, (c) the prevention of attachment of virus to the fabric, or (d) a combination of these factors.

Small numbers of viruses and bacteria in water or on environmental surfaces are important, because even a single viable microorganism is capable of producing infection in a susceptible host. This is not to say that infection will invariably produce or result in overt disease. It should also be cautioned here that the mere presence of small quantities of microorganisms in water or on environmental surfaces (such as on fabrics) does not always guarantee that transmission and infection of susceptible hosts will definitely take place. Fortunately, infection in nature is not nearly as efficient as it is in the laboratory. However, the presence of even small quantities of viruses and bacteria as environmental contaminants should always be viewed as a potential public health hazard which, under the proper conditions, could actually develop into a serious problem of epidemiologic proportion and significance.

Since it is not known to what extent pathogenic microorganisms are encountered on contaminated clothing and other fabrics which are actually processed in modern laundry and drycleaning installations in this country, and to what extent the solvents become contaminated, it would be difficult to project or extrapolate the results of these laboratory-based studies in an attempt to define the real extent of the problem as it may actually exist in the real-life situation. Such extrapolations, without objective data to back them up, would be dangerous. The quantities of microorganisms employed in these experiments are far greater than those which would be expected to be normally encountered in the natural environment. These laboratory studies do show, however, that a potential contamination problem could exist, that this might be of public health importance, and that further investigations should now be undertaken to elucidate the actual extent of microbial contamination and dissemination during commercial cleaning operations as they are presently conducted.

TOXIC FACTORS IN FOODS

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A living organism, including man, is often compared to an enormously complex machine that is functioning continuously, and carrying within itself an elaborately equipped maintenance department which makes possible necessary repairs without stopping the operation. This extraordinary maintenance department has the ability to enlarge the plant whenever necessary and desirable and even to make complete working replicas of the original machine. Obvious requirements for keeping such an unusual machine in continuous operations are:

1. Suitable fuel for energy
2. Suitable structural raw materials for maintenance, repair, expansion, or growth
3. "Operational" materials or special tools necessary to maintain proper function which the maintenance department cannot fabricate itself and which, therefore, must be supplied pre-formed.

It is clear that a characteristic of living things is dependence on external sources of nutriment. Animal life is dependent on these external sources; that is, on the environment, for the basic essentials of air (oxygen), water, and food. The ability to sustain life in the absence of oxygen is measured in minutes; in the absence of water, in days; and in the absence of food, in weeks. Even today, with our problems of air pollution becoming more pressing, oxygen is seldom a need that man must work to supply, except in his high flying planes and extra terrestrial vehicles, or in his submarine activities. Water, likewise, has been generally abundant, except in certain geographical areas, but is an essential to which we must now give more and more attention, and, for practical purposes, today we shall think of water when we mention food. Since the beginning, however, man has spent the greatest part of his time on earth working for or seeking the next meal. The vital role of food is recognized in the Scriptures, where in the book of Genesis before Adam's fall from grace, we read, "And God said: 'Behold I have given you every herb yielding seed, which is upon the face of the earth, and every tree in which is the fruit of the tree yielding seed — to you it shall be for food'. . ." Later on, after the flood, God said to Noah and his sons: "Every moving thing that liveth shall be food for you, as the green herb have I given you all."

I am not enough of a scholar to know if elsewhere in the Holy Writ there is any indication that this largesse must be accepted cautiously. I suppose some may interpret the injunction not to eat the forbidden fruit as such a warning, although I believe all would agree that this involves a considerable departure from the usual point of the story. In any event, we know that after countless generations man had learned that relatively few of the multitudinous herbs, seeds, and fruits available to him may be used for food with safety. This lesson must have been learned so painfully that many a new food has undoubtedly been accepted with great reluctance. Witness the story of the tomato, which not so long ago was regarded as a poisonous fruit. Experiences with toxic plants have probably contributed to the limited variety of foods, to the food customs and taboos that exist in different parts of the world.

At the beginning of the century, chemists had attempted to arrive at an understanding of nutritional needs by a detailed analysis of foodstuffs. The work of the pioneers of modern nutrition, such as Hopkins, McCullom, Osborne, and Mendel, had demonstrated that nutritional requirements could be determined only by a study of the performance of experimental animals during the controlled feeding of purified diets.The resulting highly selective approach to the chemistry of foodstuffs emphasized the components usually listed in food composition tables. The goal of achieving a complete analytical balance sheet for a foodstuff was set aside and has still not been attained for any single item. Preoccupation with the essential nutritional factors has tended to obscure the fact that food materials are complex mixtures of many components. We must, however, deal with each component of the diet that may be of physiological significance.

Food scientists are aware of many of the non-nutrient components of food because of the influence these have on important characteristics of food products, such as odor, flavor, texture, stability, and color. The biological scientist has been influenced in his awareness of the non-nutrient components by the fact that food may be a source of harm as well as benefit, so that those components which have a potential for harm at relatively low levels of intake and are commonly classified as poisons, are the substances he thinks of when considering nonnutrient components of foods.

It is well to remember that every food substance may be classified according to the level of intake as either physiologically inert, physiologically beneficial, or potentially hazardous. We are as much concerned with the problems caused by an excess of a food component as with the problems resulting from the deficiency of an essential nutrient. The concepts of levels of intake, although applied usually to extraneous components, is clearly illustrated by the nutrients themselves. Although it would be difficult to say at what level each is physiologically inert, there is no question that certain levels of intake are insufficient to maintain normal function. The level of nutrient requirement associated with normal health, that is, with physiological function and benefit, is well understood for most nutrients. For every nutrient there is also a level of intake that constitutes a potential hazard. The margin between the level of function and the level of hazard varies considerably with each. Everyone is familiar with the example of vitamin A in warmblooded animals, where only a tenfold increase in the usual daily requirement brings it into the range of potential hazard. One may even consider slight excesses of energy intake as constituting a potential hazard in light of the consensus that the most important problem of malnutrition in the United States today is obesity. In this case, the margin between the level of caloric intake consistent with normal physiological function, and that creating a potential hazard, is very narrow.

The nonnutrient components may be classified according to whether they are present because of the inherent genetic characteristics of the plant or animal from which the foodstuff is derived, or whether they are substances arising outside of the food itself. These latter substances can be further subdivided as to their origin:

1. Whether they have become part of the food as a result of man's activities in agriculture, food processing, storage, packaging, transportation, etc.

2. Whether they are unwanted contaminants resulting from careless handling, spoilage, or some other uncontrolled or uncontrollable circumstance.

A table of contents of a recently prepared chapter illustrates the scope of the nonnutrient components of the diet:

1. Genetically determined components of plant and animal foodstuffs other than nutrients: Cellulose, pentosans, and other poorly digestible carbohydrates, toxic factors associated with protein-rich seeds, toxic plants (on which there have been many books written), plant and animal pigments, fish toxins, and toxic algae, and naturally occurring antibiotics in foods.

2. Adventitious components of foodstuffs (food contaminants): Accumulation of soil elements; factors deriving from sanitation problems; chlorinated naphthalenes; the chick edema factor (chlorodibenso-dioxins). These are factors arising from chlorinated phenols, and may represent a much more extensive problem than we have hitherto realized. The Minamata disease, which has been well studied and identified as a problem in methylmercury poisoning, and is a fine example of environmental contamination either from man-made activities or from geological sources whereby certain fish species, particularly those that tend to grow older and larger, accumulate levels of methylmercury that may represent a definite hazard to health of people. The polychlorinated biphenyls are an example of a chemical industrial contaminant that, because it was not or cannot be properly contained, has found its way through the environment into the food chain. At present, it constitutes a problem only of accidental sporadic contaminations. Hopefully, we have caught it in time so that it does not become a universal background problem threatening health of man and animals.

3. Components resulting from technology: Food additives; agricultural chemical residues, and toxic substances from food processing. These arise in various situations, as for example, the interaction of the solvent and the protein during extraction procedures, such as the interaction of trichloroethylene with soybean oil meal to form a toxic sulfur amino acid; the reaction of ethylene oxide to destroy essential nutrients and also to form ethylene chlorohydrin which has potential for harm; the production of methionine sulfoximine by the now obsolete process of bleaching flour with nitrogen trichloride; changes in fat and other lipid constituents in foods by commercial processing, storage, and by home cooking practices; the production of polycyclic hydrocarbons, some of which are potent carcinogens, in the smoking of foods and the broiling of meats; products of the browning reaction; oxidative changes in fats; and the production of metabolites of mold, and the presence of molds themselves.

Foods to which we must look for a supply of the defined nutrients that are essential for health and life consist of much more than the 50 to 100 nutrient chemicals. Our foodstuffs are derived from plant and animal life. If we were to list all of the known chemical substances of which these are comprised, we would have a formidable catalog of complex chemical structures that would make the list of 2,000 to 3,000 food additives and several hundred pesticides (which have received so much concern) look like a kindergarten exercise as compared with a multivolume treatise. We are fully aware that in most cases we have not yet scratched the surface with regard to knowing the nature of minor components that are present in our foodstuffs, for in no single case do we as yet have a complete balance sheet. In this connection, it is important to point out that about 10 years ago we were still in the era where the analytical chemist felt very good about his accomplishment of being able to measure with a sensitivity of parts per million. Except for very sensitive bioassays, parts per billion were an impossibility for most chemical procedures, but with the advent of various forms of chromatography, including gas-liquid chromatography, chemists today feel quite confident about their capability of measuring in the range of parts per billion. And today we are on the verge of a new era in analytical chemistry where, by combining immuno-chemistry with radioactivity measurements or electron-spin-resonance measurements, we can think confidently of analytical sensitivities down to parts per trillion. It is important to remember that these are just not idle curiosities; that from the point of view of toxicity, these kind of sensitivities are often important. In considering how much of a substance or an impurity is present, the criterion cannot be purity as defined by the old cliche of 99.4 percent pure. In some cases, this degree of purity is grossly inadequate and sometimes it is unnecessary. The degree of purity depends entirely on the specific impurity we are concerned about. The question is not how pure, but is it pure enough? And for some things, we believe analytical sensitivity in parts per trillion is not asking for too much. Priority requirements should be based on toxicological criteria rather than available analytical capability.

Many of our established methods of food preservation and food processing, such as milling, baking, dehydration, fermentation, brining, and sugaring and a variety of heat treatments, date back to prehistoric times, and their safety has been tested in the "crucible of human experience," not always, however, definitively, and with finality. For example, in retrospect, milling of grains with the resulting widespread incidence of beri-brei and pellagra, could hardly be considered to have been a wholesome practice. We should remember that the water, the foodstuffs, and food processing procedures that we accept today without reservation as to their safety and wholesomeness, enjoy this acceptance entirely on the basis of short-term observations. We still do not know how to reliably associate effects, that is, diseases seen in the human population, with causes that have acted early in the life of an individual and are now gone, or which have been acting subtly by slow accumulation of a deleterious factor or effect. The absence of acute effects is probably the primary, if not the only basis for acceptance of all our foods that have been "tested in the crucible of human experience."

Legislative developments in recent years brought about by an increase in popular awareness of the large numbers of extraneous substances in foods, such as pesticide residues and food additives, have compelled biological scientists of many disciplines, to be concerned with these particular food components, so much so that there has been a strong tendency to overlook those substances that are naturally present.

I have dwelt at some length on the complexity of the chemical composition of our foodstuffs and the fact that we have a great deal still to learn about this. You may be wondering what all this has to do with the toxic factors in foods that I am supposed to be talking about. In considering this question, it is important to remember that toxic effects are always related to the level of intake and that hazard is associated only with the presence of the substance in excess. The determination of how much constitutes excess is what toxicology is all about. The difficulty of defining a toxic factor is exemplified by the spice, nutmeg. In small quantities it is used as a condiment, apparently without hazard but, in large amounts, such as a whole nutmeg, it is reported to show very poisonous properties. The toxicity, or capability of a substance to do harm is usually expressed as the LD_{50}. The commonly used terms describing toxicity, such as extremely toxic and highly toxic, are associated with rat LD_{50}'s, varying from 1 mg. or less per kg. for extremely toxic, to 5 g./kg. for slightly toxic.

Anything with an LD_{50} of 15 g./kg. or more is considered to be relatively harmless. LD_{50} values, however, are not very useful as indicators of chronic toxicity or of carcenogenicity. For foods we are concerned with chronic effects primarily, since long-term or lifetime ingestion is involved. Acute effects are easier to identify and to avoid, whereas chronic toxic effects such as carcinogenicity, are much more difficult to evaluate. Obviously, a major concern in any consideration of toxic factors in foodstuffs must be with subtle long-term effects. Man has learned to avoid dangerous foodstuffs which produce acute effects or through processing to render them useful and safe. However, the relation of the intake of minor food components to old and continuing problems, such as cancer, congenital defects, arthritis, and heart disease, has only begun to be explored. We should remember that these problems have been with us for a long time before the present technological era and before concern with food additives and environmental contaminants had impressed itself on the public consciousness.

There is a factor of approximately 10 million between extremes of potency of toxic substances. For example, some are toxic at parts per hundred of the diet, whereas others are toxic at parts per billion. Therefore, in assessing a potential hazard, we must know its relative toxicity as well as the extent of the presence of the substance in air, water, food, and soil. And since certain toxicological problems relate to the slow accumulation of a substance in the body over many years until it reaches a level in one tissue or organ that interferes with normal function, we should learn early, in addition to the ability to cause harm, whether or not a substance has a tendency to accumulate in tissues, and what its residence time in the body may be.

In a discussion last year on food safety, Dr. Wodicka, Director of the Bureau of Foods, discussed six specific hazards associated with foods in what he considered to be their order of decreasing importance. First, and most important, on the list, he considered to be the microbiological hazard; second, the hazard of malnutrition; third, the hazards that derive from environmental contaminants, and following in order, hazards from naturally occurring toxins in plants and animal life, then pesticides, and last the hazards from food additives. When I was here 8 years ago, I said "a discussion of toxic factors would be incomplete without mention of sanitation. The problem of contamination of some of our foodstuffs by salmonellae exemplified the importance of this factor. That food and feed, particularly when contaminated by filth and insects, can serve as efficient vectors for disease is well known, and is one of the first possibilities to be considered when investigation of a new epizootic or epidemic disease is begun." It is probably true today that more people are affected in this country by microbiological contamination of food than there are by any other impairment of food safety. You have heard Dr. Ayres discuss this in depth. I would like to add merely that poisoning caused by Clostridium botulinum, which is widely known and dreaded because, although not common, is frequently fatal when it does occur, is under good control primarily because of the successful development and control of food processing. All our cured meat products, which represents a very substantial portion of the total meat supply, apparently have been protected against botulinum hazard by the use of nitrite as an ingredient of the curing mixture to an extent greater than we had realized.

I also said at that time: "There would be no point in reviewing here the long list of pesticides and food additives that are used in agriculture and food production. The safe use of each of these materials is well understood and controlled by law, so that the residues remaining in accordance with the established tolerance should not represent a hazard to man or domestic animals. However, many of these substances are toxic, and continuous vigilance must be maintained to assure only established safe residue levels."

A word about safety evaluation is in order. Evaluation of the safety of a substance must be based on uncovering its potential to cause injury and on acquiring sufficient data to warrant a conclusion that, under conditions of proposed use, the intake is so low, in relation to the toxic dose, that there is a practical certainty no harm will result.

Studies in groups of animals or people have inherent limitations imposed by the limited number of experimental subjects available, and the fact that they are all not equally susceptible to every type of insult or injury. Ideally, we should like to have an experimental animal that has high susceptibility to every possible type of injury and in which the functional, morphological, and biochemical abnormalities are comparable with those observed in humans. Furthermore, the experiment should be designed to detect every conceivable type of toxic effect in every tissue and organ. Obviously, no one has discovered this ideal animal or test situation. The toxicologist, therefore, is not surprised to discover that studies made years earlier are sometimes lacking in one regard or another and that earlier conclusions must be modified. The ordinary consumer or even the sophisticated or educated one, often has the notion that safety evaluation involves simple test procedures that are capable of giving absolute answers. I have not yet learned how to explain the difficulties and intricacies of safety evaluation to a consumer who insists that practical certainty of safety is not enough — that there must be absolute safety. It may be that as we apply a new test we may see a new effect that has not been seen before.This does not necessarily mean that the effect is deleterious or that a hazard is posed at the level at which the substance is present in food. But sometimes this may be the case. An old and familiar substance may have to be relegated to the scrap heat or controlled to much lower levels of intake than formerly.

It is well known either from experimental studies or long experience that many of the multitudinous and varied physiologically active agents that may be present in food exert no deleterious effects because of their relatively low dietary intake. However, it is always necessary to be alert to the potential hazards that are inherent in the intake of substances in excess. It is, therefore, necessary for us to know the nature of the components of food, their potential for producing harmful effects, as well as their level in the daily diet. It is important to point out two aspects of food processing which represent potential hazards. First, it should be remembered that many of the processes result in separation of desired ingredients from raw materials and will also result in removal of components that may have protective actions. The removal of micronutrients by milling is a classic example. Some have postulated the presence of protective dietary factors different from the known nutrient, to explain apparently protective effects of commercial type of stock rations composed of practical dietary ingredients as compared with the response of animals on highly purified diets. Secondly, and for our present discussion of much greater concern, are the potential hazards that result from concentration. For instance, the separation of oils from oilseeds represents a concentration of potentially deleterious effects from excess consumption of fat or specific fatty acids. We must always be alert to the possibility that a minor component of food may be concentrated during the preparation of a new food product to the point where it represents a hazard to health. In this vein, vitamin A is concentrated by extraction or through synthetic processes, the potential for excessive levels in food, whether by accident or design, is a real hazard to be avoided. The preparation of fatty acids by distillation of hydrolyzed oils resulted in concentration of chick edema factor from nondetectable levels to highly toxic concentrations.

Remarkable physiological effects can be produced by extremely small amounts of certain substances. The story of the vitamins illustrates the success possible in the difficult task of detecting, concentrating, and isolating in pure form, components present in only parts per million or parts per billion. The key is always some sort of bioassay or a test that correlates with the bioassay. The same general approach has been used in the isolation and identification of minor, nonnutrient components that exert undesirable effects. For example, the first evidence that the toxic principle in toxic wheat was selenium was obtained by feeding the ash of the wheat to rats. When the same toxic effects as with the original toxic wheat was obtained, and the comparison of the inorganic elements in the toxic wheat with samples of nontoxic wheat revealed the high selenium content, the identification was possible. The aflatoxin story, its discovery, concentration, and identification is a variation on the same theme. The implication several years ago that cottonseed meal was a causative agent in trout hepatoma brought to the fore the question of how to proceed to establish the wholesomeness of the basic food.

The problems of evaluating the safety of a food are somewhat different from the evaluation of a pure or relatively homogeneous substance that can be fed at many times the level of its proposed use. In addition to evaluating nutritive value, which is a relatively straightforward matter, it is important to determine whether any toxic components, adventitious or otherwise, are present and whether, considering the conditions of use, they will present a hazard to health. It would seem logical to make use of our accumulated experience with the isolation and identification of many naturally occurring physiologically active substances in the solution of this latter problem. Instead of depending entirely on responses observed during feeding of the food itself, why not administer a series of extracts and concentrates? In this way, we would have the advantage of increasing doses and studying the biological response, if any, just as we do in the case of chemical additives. This approach should yield great dividends in knowledge and assurance of safety.

After countless generations, man has learned that relatively few of the multitudinous herbs, seeds, and fruits may be used for food with safety. Noxious weeds were recognized in Biblical times, when sowing of tares was regarded as an unfriendly act, as is dramatized for us in the parable from St. Matthew (xiii 25) "but while man slept, his enemy came and sowed tares among the wheat." It has been pointed out that Nature, while generously supplying our daily food, has kept toxicologists busy since ancient times investigating such naturally-occurring toxicant agents as hemlock; the poisons in certain mushrooms; the atropine in some members of the nightshade family; the digitoxins of foxglove; the ricins of the castor bean; the cyanogenetic glycosides in fruitseeds; the gossypol in cottonseed; the goitrogens in cabbage, kale and rutabagas; the alkaloids of the locoweed; and the toxins of certain fish and shellfish. Man has learned to avoid dangerous foodstuffs or by processing to render them useful and safe.

The myriad nonnutrient components of food emphasize that their mere presence does not make food unsafe. It is their presence "in excess" that is hazardous. The Pesticide Amendment in 1954 and the Food Additive Amendment in 1957 established the principle that "toxic substances" may be legally used in food production and may be present in foods at "safe" levels. The principles and practices of safety evaluation have been discussed in many places. Regarding most of the substances present in our common foodstuffs, we tend to rely on centuries of human experience to assure us of the safety of foods which contain them. However, until we understand completely the etiology of all the so-called metabolic and degenerative diseases, we should keep an open mind about the potentialities for harm, as well as benefits inherent in the composition of even our most important foodstuffs.

SESSION III: "Safety" — James E. Halpin, Agricultural Experiment Station, Clemson University, Clemson, S.C., Chairman

ENFORCEMENT BY THE FEDERAL TRADE COMMISSION

by
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In view of its expanded responsibilities under the Flammable Fabrics Act, the Federal Trade Commission has adopted the following policy for enforcement of the Flammable Fabrics Act to insure maximum protection of the public through voluntary, compulsory, and other procedures.

ENFORCEMENT POLICY

As a public safety statute, the Flammable Fabrics Act must be enforced vigorously, effectively, and without compromise. Every reasonable means for enforcement must be promptly employed, every alternative must be fully explored, and every available resource must be brought to bear. The efforts of the Federal Trade Commission to enforce the Act must be unyielding, unequivocal, aimed solely to discharge with optimum effectiveness its responsibilities under the Act to protect the public from the threat to health, life, and property that inheres in potentially dangerous flammable products covered by the Act.

ENFORCEMENT OBJECTIVES

To acomplish the foregoing enforcement policy, resolute and expeditious enforcement of the Act will be premised upon the following objectives:

1. To prevent the entry into the channels of commerce of any products, fabrics, or related material, hereinafter referred to as "flammable fabrics," which fail the applicable flammability standards set pursuant to the Flammable Fabrics Act (15 U.S.C. 1191);
2. Immediately upon discovery of flammable fabrics, to stop, by the most effective means available, the sale and distribution of such fabrics which have reached the channels of commerce;
3. To notify the consuming public and all other parties in the channels of commerce of the potential danger of such fabrics;
4. To cause all flammable fabrics to be taken out of the channels of commerce by any means available by law; and
5. To prevent the re-entry of flammable fabrics into the channels of commerce.

ENFORCEMENT PROCEDURE

Voluntary Procedures

In affecting immediate removal of flammable fabrics from commerce, and alerting all levels of distribution of the hazard involved in such fabrics, the Commission seeks and encourages the voluntary cooperation and assistance of affected parties in terminating the sale and distribution and obtaining removal of the flammable fabric from commerce.

In specific instances where products have been tested and failed, the staff is directed by the Commission to proceed as follows:

The manufacturer, importer, distributor, or retailer of the flammable fabric, or any combination of such persons, is to be contacted immediately and notified orally of the results of the applicable flammability test. Written confirmation of such notice will also be made. Such person or persons will be asked to voluntarily (1) stop the sale and distribution of the flammable fabric, (2) furnish to the Commission Staff a list of customers who have purchased the product, (3) notify those customers of the test results, (4) seek removal and/or recall of the flammable fabric from commerce, and (5) supply inventory data showing the amount of the flammable fabric purchased on hand and in the channels of commerce. Except for good cause shown, these voluntary procedures must be completed within 5 days of notification by the Commission Staff.

Such person or persons voluntarily complying with the foregoing procedures will be provided the opportunity to execute a consent order agreement, pursuant to Section 2.14 of the Commission's Rules. This settlement offer, containing an agreed-upon complaint and order incorporating among other things the foregoing procedures, will be forwarded to the Commission for final disposition in accordance with its Rules of Practice. This is pursuant to the policy of the Commission which is to require issuance of a formal cease and desist order in every case where a violation of the Flammable Fabrics Act is found and not to accept Assurances of Voluntary Compliance in these cases. Such person or persons will be informed that a press release will issue pursuant to this voluntary agreement announcing that advice regarding the distributors, retailers, and other handlers of the product in question can be secured from the Commission by the use of the toll-free number listed in the press release or by writing to the Assistant Director for Textiles and Furs. If the party complies and the Commission accepts the proffered agreement, the press release will be issued immediately.

Such person or persons will be informed that if he fails to comply voluntarily with the procedures set forth above, the staff will proceed immediately to utilize all appropriate compulsory processes available to the staff to accomplish its enforcement objectives and to protect the public interest. Such person or persons will be informed as to the nature of those compulsory processes.

Compulsory Processes

If any person or persons reject a Section 2.14 settlement or refuse to cooperate entirely in each of the above-described voluntary procedures, he will be orally informed, with this information confirmed in a subsequent letter, that the staff intends to recommend to the Commission the issuance of a complaint under Part III of the Rules, and the staff will recommend that the Commission commence proceedings in the District Court seeking all relevant information or data previously withheld, a court injunction against further distribution of the product, and seizure by Federal marshalls of the product. In such event, a press release shall issue announcing issuance of the Commission's administrative complaint instituting formal proceedings and the commencement of proceedings in the District Court. The staff will further advise such person or persons that these enforcement procedures also will be taken, where appropriate, against the distributors or retailers of the product.

Other Enforcement Procedures

In-house test results of such person or persons indicating that offending products meet flammability standards will not be sufficient to suspend implementation of the above enforcement procedure so long as the test, relied upon by the Commission, performed in accordance with the applicable flammability standard, shows failure of the product. If reliable tests by independent laboratories ordered by the party show that the product meets flammability standards, the staff will confirm the results of the tests previously relied upon by the Commission before determining whether to pursue the foregoing procedure.

The Commission as an adjunct to the above enforcement policy has set up a special Flammable Fabrics Information Center designed to provide consumers with information concerning Commission action involving enforcement of the Flammable Fabrics Act.

The Commission will continue to issue news releases that fully describe flammable products but will no longer include the customers lists in the releases.

Instead, information about the names of retailers and other customers, the regions and cities in which they are located, and other information that consumers may seek with respect to flammable products will henceforth be provided by the new Flammable Fabrics Information Center to anyone who wishes to call the following toll-free number between 9:00 a.m. and 5:00 p.m. Eastern Time, Monday through Friday: (800) 424-8589.

The same information may also be obtained by writing:

Division of Textiles & Furs
Federal Trade Commission
Washington, D.C. 20580

FLAME RETARDANT BEDDING PRODUCTS[1]

by
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ABSTRACT

On September 9, 1971, the Department of Commerce announced a proposed standard to measure the flammability of mattresses. The standard employs lighted cigarettes. The use of cigarettes to measure flammability introduces problems significantly different than those associated by classical theory with flame retardance. Where the cigarette is used as an ignition source, the major factor becomes one of heat

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[1] This research is being carried out under a cooperative agreement between the U.S. Department of Agriculture, The National Cotton Batting Institute, and Cotton Incorporated.

transfer rather than chemical interference with the course of combustion. Smoldering combustion has been cigarette-induced in cotton batting that passes the vertical flame test, and such combustion is self-sustaining in atmospheres containing as little as 1 percent oxygen. Recent research indicates that heat dissipation techniques offer promising ways of inhibiting ignition of mattresses by cigarettes.

INTRODUCTION

On September 9, 1971, the Department of Commerce published in the Federal Register a proposed standard for the Flammability of Mattresses. As had been anticipated, the standard promulgated involves the use of three lighted cigarettes to determine flammability. Bare mattresses and mattresses covered with two sheets are tested. No provisions are made for the use of an open flame, even though a study made by the Southwest Research Institute at San Antonio, Tex., for the National Bureau of Standards states that mattress fires initiated by methenamine tablets, or by matches, ignited earlier and developed hazardous conditions more rapidly than did fires caused by cigarettes.

The manner in which cellulosic materials burn depends upon many factors: the source of ignition, the heat transfer environment, the oxygen supply, and the presence or absence of foreign materials such as finishing agents or natural contaminants such as oils, pectins, or waxes. These, in combination with the physical structure of the cellulosic product, influence the path of combustion to either flaming or smoldering. In general, flaming combustion involves rapid oxidation in a gas-phase reaction. Smoldering combustion is a direct reaction wherein a solid combines with gaseous oxygen. The term "smoldering combustion" has been described in many ways. Flameless combustion, glowing combustion, afterglow, and solid state combustion are frequently used in the literature to describe thermal degradation in the absence of an open flame.

Flameless combustion, or smoldering combustion, is sometimes mistakenly considered as a sequel to flaming combustion with the term afterglow applied to the phenomenon. It is important to recognize that flaming combustion is neither a prerequisite nor a sufficient condition for the occurrence of smoldering combustion.

From a chemical standpoint, flaming combustion involves the rapid oxidation of flammable gaseous pyrolysis products. Classical flame retardant theory envisions an arrangement of the chemical reactions of pyrolysis so that fewer flammable volatiles and more carbonaceous residue is produced. This approach to flame retardance, however, makes the problem of preventing smoldering combustion more difficult, because in practice it results in the production of increased amounts of carbonaceous fuel for the solid-state reaction.

The classical approach works acceptably well for cotton fabrics in a vertical test. However, when flame retardants are used to suppress smoldering or flowing combustion, serious problems are encountered in products such as cotton batting where ignition is achieved by a flameless source. This has been found to be associated with the heat transfer characteristics of fabric vs. cotton batting under certain very specific conditions. For example, it has been observed that cotton batting products that readily pass a vertical flame test, under some conditions, can be brought to a self-sustaining smoldering combustion using a cigarette as a source of ignition. Such a finding seems to be an anomaly since the flame from a bunsen burner in the vertical flame test is approximately 1,600° F. and considerably larger as a source of heat flux than a cigarette which burns at only about 900°. It is important to recognize the differences inherent to the respective tests. In the cigarette test the total energy supplied is 8 to 10 times greater than is provided by the vertical open flame test. This difference is attributable mainly to exposure time to 12 seconds in the vertical open flame test vs. 20 minutes in the cigarette test. In the vertical flame test the sample can readily conduct, convect, and radiate heat to the surroundings because of its orientation during test. In the cigarette test, the orientation of the batting is such that the insulative characteristics of the product are brought into play. As a result, thermal energy is absorbed and stored until a temperature of about 750° is reached. Smoldering combustion ensures. This combustion becomes self-sustaining because the insulative characteristics of the batting prevents the radiation, convection, or conduction of heat energy. The smoldering combustion reaction generates heat faster than it is removed from the system causing the thermal energy to initiate the smoldering combustion.

In mattresses and upholstered furniture containing cotton batting, combustion is usually of the smoldering type. Flaming, if it occurs at all, is merely an antecedent which provides sufficient thermal energy to initiate the smoldering combustion.

It has been shown by differential thermal analysis techniques that smoldering combustion is self-sustaining in atmospheres containing only 1 percent oxygen so long as the temperature is above 750° F. Smoldering combustion ceases when the flame retardant treated batting is exposed to conditions under which the energy is dissipated, and the internal temperature falls below 750°.

A word of caution is needed. The analysis made above should not be construed as meaning that flame retardants have no part in the development of mattresses that will pass the proposed cigarette test for flammability. The presence of flame retardants significantly slows the pyrolysis reaction. However, increasing the concentration of flame retardant above that required to pass the vertical flame test does not enhance smolder resistance except when the agents are used at impractical levels.

Construction of the mattress in heat dissipation techniques seem much more promising as a means of preventing the initiation of smoldering combustion than does the use of excessive amounts of flame retardants.

A number of tickings have been backcoated with: (1) aluminum powder, (2) alumina trihydrate, (3) blowing agents, (4) thermoplastic resins, and (5) intumescing agents.

Sample mattresses have been constructed of flame retardant batting covered with flame retardant, backcoated cotton ticking which successfully passed the cigarette test. Effective backcoatings are based upon vinyl chloride or acrylic polymers, usually at about a 10 percent add-on by weight of the ticking. In some cases it was possible to prevent ignition when the backcoating contained only 1.25 percent of aluminum powder.

Thermodynamic and heat transfer studies indicate that the add-on of aluminum trihydrate to absorb the energy being supplied by a cigarette would have to be above 0.5 $gms/cm.^2$. This follows from the fact that only 240 of the 480 calories per gram needed in the dehydration reaction are available in the time-temperature cycle obatined from a cigarette.

Blowing agents appear to be effective in dissipating the heat. The price of these materials, however, makes their use in the backcoating of the ticking rather doubtful.

Some of the vinyl chloride and acrylic thermoplastic resins individually appear to be helpful in dissipating the heat from cigarettes. Larger add-ons are needed than when aluminum or alumina trihydrate are used with them. More study is needed to clarify the economics.

Intumescing agents have been explored. Some of these can be compounded at rather low prices and the add-ons needed do not seem to be excessive. However, when used in backcoating materials there is some evidence that the pressure of the ticking on the underlaying batting may minimize the tendency of the intumescing agent to expand when heated.

All of the backcoating additives mentioned have been applied with thermoplastic film-forming latices. Most useful have been a phosphorylated vinyl chloride, a carboxylic acid modified ethylene polyvinyl chloride, and an acrylic copolymer. The usual add-on needed has been between 8 and 10 percent by weight of the ticking.

It is recognized that the use of such backcoatings will reduce the air permeability of the ticking, usually by 70 to 90 percent. It has yet to be determined if this is too great a loss in permeability to be tolerated without affecting the comfort of a mattress. There are, however, indications that air permeability affects the ability of the products to inhibit the ignition by cigarettes. Reduction of air permeability seems essential to deter ignition. In fact only when the air permeability of the ticking was below 2 $c.f.m./ft.^2$ or less did mattresses survive attempts at cigarette ignition.

Nonfilm-forming latexes of the vinyl chloride type are now being investigated by evaluating the binding of the heat dissipator with such nonfilm-formers. These latexes should help to explain the part played by air permeability in inhibiting ignition.

One of the most vulnerable locations on a mattress is the rolled or taped edge. Ignition (where cigarettes are placed in the valley formed by the roll or tape and the body of the mattress) can be prevented by the inclusion of two strands of single solid No. 14 electrical wire or its equivalent in stranded wire. More work is needed to determine the minumum amount of wire that will prevent ignition. Following the above premise it has been demonstrated that the tendency to ignite can be minimized by the use of tapes that contain metallic yarns.

The National Bureau of Standards has indicated that ignition of cotton mattresses by cigarettes can be prevented by the use of ¼ — to ½-inch of polyurethane foam beneath the ticking. They believe the foam melts and forms a void which then insulates the cotton from the heat from the cigarette. Our results do not confirm their findings.

During the recent interlaboratory testing of mattresses to establish the reproducibility of the cigarette test, some solid foam mattresses failed the standard. Vulnerable ignition points were found where the foam is quilted to cotton ticking. Likewise the foam seems to offer little or no protection from ignition at the rolled or tape edges.

In summary, some progress has been made toward the development of mattresses that will pass the cigarette test. Data to date point to the need for some changes in the components other than the batting to achieve a composite structure that will pass the cigarette test. What this means is that the application of flame retardants to the ticking and to the batting will not in themselves suffice to achieve a mattress that resists ignition by burning cigarettes.

Flame retardance treatments for the batting increase the temperature at which smoldering combustion is initiated. This is desirable, and probably will be essential if mattresses that pass the cigarette test are to be manufactured on a day-to-day basis.

The ticking probably will have to be modified. It should function to dissipate the heat from a cigarette so that the temperature of the treated cotton batting below the ticking is prevented from reaching the critical 750° F. at which smoldering combustion occurs. Current data indicate that heat dissipation can be achieved by backcoating the ticking with suitable thermoplastic resins. It is interesting to note that a number of ticking mills have already resorted to backcoating as a means of maintaining or reducing the cost of cotton ticking.

Other changes such as the incorporation of metal wire or metallic yarns into the taped or rolled edges minimizes the potential for ignition at this particularly easily ignited part of the mattress.

SKIN REACTIONS TO TEXTILE FINISHING COMPOUNDS (SUMMARY)

by
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The use of fabric finishes in the manufacture of clothing textiles has become widespread, and the various compounds which are used may occasionally cause an allergic contact dermatitis in the wearer. The formaldehyde resins have been responsible for most of the reactions of this type which have been reported[1].

The physician may first suspect the presence of a clothing dermatitis when a patient is seen with a dermatitis involving those areas of the body which are warm, moist, and in close contact with clothing fabrics. These areas of involvement include the sides of the neck, the underarm areas, the creases of the arms and knees, the belt line area, and the buttocks and the inner parts of the thighs. To establish that the clothing dermatitis is caused by the formaldehyde resins, which may be contained in the fabrics, the following criteria should be fulfilled:[2]

1. The suspected fabric should give a positive chemical test for formaldehyde.
2. The patient should have a positive patch test reaction to 2 percent formalin.
3. Those fabrics giving a positive chemical test for formaldehyde should produce a positive patch test reaction in the patient.
4. The wearing of the fabric giving positive tests for formaldehyde should produce a recurrence of the dermatitis in the patient.

Two chemical test methods are commonly used to detect the presence of formaldehyde in textile samples. The methods are the Schiff's reagent test[3] and a modification of Eegriwew's test for formaldehyde[4]. The Schiff's reagent test is done as follows:

1. Sample of material (approx. 1x4 cm.)
2. Immerse in 5 ml. O.1 N HCl
3. Heat in water bath (68 to 80° C.) 10 min.
4. Add 5 drops of Schiff's reagent
5. A violet color developing in the course of a few minutes indicates the presence of free aldehyde released from the fabric and in textiles this will almost certainly represent formaldehyde.

The Eegriewe's test is performed as follows:

1. Sample of fabric (approx. 1x4 cm.)
2. Immerse in 5 ml. 1 percent H_2SO_4
3. Let stand for 5 minutes
4. Add 1 drop of solution to 2 ml. 75 percent H_2SO_4, add a few grains of chromotropic acid
5. Heat mixture over flame
6. A positive reaction consists of an intense reddish-purple color.

During the past 6 years a total of 10 patients with clothing dermatitis caused by allergic contact sensitization to formaldehyde resins contained in clothing textiles have been seen in the Department of Dermatology at Louisiana State

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[1] Quinn, S. E., and Kennedy, C. B. Contact dermatitis due to formaldehyde in clothing textiles. Jour. Amer. Med. Ass. 194: 593-596. 1965.

[2] Fisher, A. A., Kanof, N. B., abd Biondi, B. S. Free formaldehyde in textiles and paper; clinical significance. Arch. Dermatol. 86: 753-756. 1962.

[3] Wereide, J. Formaldehyde as a contact allergen in textiles: formaldehyde clothing dermatitis. A ten year period in-patient series (1953-1962). Acta Allergol. 19: 351-352. 1964.

[4] Marcussen, P. B. Dermatitis caused by formaldehyde resins in textiles. Dermatologica 125: 101-111. 1962.

University School of Medicine. The total number of patients involved with this condition is not large but because the use of formaldehyde resins has become so widespread in the textile industry, it is very difficult for patients who have this condition to find suitable clothing. The chemical tests described above have been of considerable usefulness in screening fabrics for possible use by patients with this condition.

REMOVAL OF EXCESS FORMALDEHYDE FROM TREATED TEXTILE PRODUCTS

by
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Some of the serious problems which occur when those allergic to formaldehyde are exposed to durable press textile fabrics which give off formaldehyde have been detailed by O'Quinn (8). The present paper describes the more general problem of free formaldehyde on treated textiles and some of the methods of reducing or eliminating this hazard.

Free formaldehyde is not chemically reacted with the fabric. Thus, it readily volatilizes into the air and provides a source of irritation to workers who must handle the fabric. This industrial air pollution creates both a health hazard and an annoyance. Workers in garment plants have become more militant lately in their demands that the formaldehyde air pollution problem be overcome.

In the durable press process, chemical agents are used which crosslink or "set" the fibers (very much like setting a permanent wave in hair) so that the fabric will retain its shape after laundering. Thus, after laundering the fabrics dry smoothly and pressed-in creases are retained. Although many crosslinking agents have been investigated, almost without exception, the ones used are based on formaldehyde and are the source of the formaldehyde vapors.

Two processes are used in the manufacture of durable press garments, the pre-cure and the post-cure. The pre-cure process is employed mainly for light-weight fabrics, such as those used in shirts, sheets, and dresses. Yard goods are treated with the crosslinking chemical at the textile finishing plant and the fabric is cured at a high temperature — say 320° F. for a few minutes to set the fibers. The high temperature volatalizes much of the excess formaldehyde which is blown away from the fabric in the oven. However, a certain amount of free formaldehyde is left in the fabric. This residual formaldehyde is the irritating odor most people notice in a yard goods retail store. At the Southern Regional Research Laboratory (SRRL), we found that washing the cloth removes free formaldehyde and reduces release of formaldehyde to the low level of 1,000 parts per million (p.p.m.) or about 0.1 percent (13). Unfortunately, the finisher is not willing to pay the half to one cent per yard it would cost to remove this formaldehyde by washing and drying the fabric.

The second durable press process is known as the post-cure process. This is generally used for the heavier cottons and cotton/polyester blends which are designed, for example, for the manufacture of men's trousers. Trousers require neatly pressed creases for good appearance. If the trousers were made from pre-cured fabric, they would resist pressed-in creases just as they resist wrinkles. In the post-cure process, this difficulty is circumvented by treating the goods with the crosslinking agent and drying the fabric but not curing it. That is, the fabric has the agent present in it, but the agent has not reacted with the fibers. Then the garment is cut, sewn, and pressed. After that, the garment is passed through a hot oven for about 10 minutes to complete the process. Thus, creases are cured in place at the same time that the smooth areas are cured.

In the post-cure process, the formaldehyde-based agent is dried on the cloth, but not reacted with it until the final step of the manufacturing process. Therefore, fabric with a post-cure treatment has more free formaldehyde than cured fabric and it releases more obnoxious formaldehyde fumes during the time the fabric is being cut, sewn, and pressed.

In all fairness to the industry, it should be pointed out that efforts have been made to provide good ventilation and there have been no reports of serious health damage. On the other hand, there is no doubt of the existence of irritating and uncomfortable working conditions. Also, the less tangible question of possible health hazard resulting from long time exposure to small amounts of formaldehyde remains to be settled.

Toxicology

It is rather surprising that more is not known about the effects of formaldehyde on the human body. Formaldehyde cannot really be considered to be a "deadly" poison. Deaths from ingestion of concentrated liquid solutions are extremely rare (7). In the vapor state, formaldehyde is one of those compounds which is so irritating that it acts as its own warning agent. Thus, the odor becomes intolerable long before the lethal concentration is reached. On the other hand, formaldehyde is insidious. Because it is very soluble in water, it dissolves very quickly in the mucous membranes of the nose, upper respiratory tract and the eyes, thus concentrating the irritation rather than spreading through the body. It combines with the proteins and irreversibly alters them. An interesting point is that blonds seem to be more severely affected than brunettes (5). It is well known, of course, that exposure to a small amount of formaldehyde will greatly reduce, temporarily, the sensitivity of the olfactory nerves.

In table I is a compilation of the effects on humans of formaldehyde vapor concentrations which are pertinent to this discussion. An interesting part of this table is the implied disagreement between references. That is, in 1944, the maximum allowable concentration for an 8-hour-day was 10 p.p.m. or just half the "unendurable" concentration. In 1961, this figure was halved to 5 p.p.m., which is also the "lowest concentration causing throat irritation."

The question then becomes one of the effect of long-term exposure. Hamilton quotes A. Brunnthaler, writing in 1914, as insisting that formaldehyde has a cumulative effect which may result in obstinate dermatitis or chronic bronchitis (4).

Table 1. Effect on humans of various concentrations of formaldehyde vapors

Concentration[1] parts of formaldehyde per million parts of air	Effect	Reference
0.8 to 1.0	Least detectable concentration	(17)
5.0	Lowest concentration causing throat irritation	(17)
5.0	"Threshold Limit" (Defined as concentration which would have no effect on worker throughout normal work day[2])	(17)
10	"Maximum allowable concentration in air during 8-hour day"[3]	(17)
20	"Unendurable"[4] Causes eye irritation	(3, 5, 17)
650	3-hour exposure damages lungs 8-hour exposure may result in pneumonia	(5)
4300	Has corrosive effect on eyes	(17)

Reduction in Formaldehyde Release

Although the question of health hazard has not been resolved, it has now become necessary for the amount of formaldehyde released from post-cure cloth to be reduced. Until early 1971, "sensitized cloth" — that is, cloth to be cured after the garment was made — released an average of 4,000 to 5,000 p.p.m. of formaldehyde. This was attained, mainly, with only one agent-dimethylol dihydroxethyleneurea (DMDHEU). Other agents had already been found to be intolerable for use without special precautions. However, in 1971, one of the large garment makers decreed that the amount of formaldehyde released must be reduced by one-half — that is they would not purchase fabric releasing more than 2,000 p.p.m. (6). This lead was soon followed by several other companies.

The procedure used to determine formaldehyde release has been criticized as being unrealistic, variable in results, and inaccurate. However, fabric now used for garments by certain companies must meet the test. The test was established by the American Association of Textile Chemists and Colorists (1) based on research conducted as this Laboratory in 1960 (9) In the test, a weighed sample of fabric is suspended over an amount of distilled water in a sealed jar. The jar is placed in an oven and held at 49° C. for 20 hours. The formaldehyde released from the fabric and absorbed in the water is determined quantitatively. The amount of formaldehyde released is expressed in parts formaldehyde per million parts of fabric.

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[1] 10 p.p.m. is approximately 0.012 mg. per liter of air at 25° C. and 760 mm. of mercury.
[2] According to American Conference of Industrial Hygienists (1961).
[3] According to American Standards Association (1944).
[4] As stated by Haggard (3).

Even at 4,000 p.p.m., it would seem that the formaldehyde in the cloth should not be excessive. A simple calculation indicates that a volume of air only 800 times that of the cloth would result only in 5 p.p.m. It would seem reasonable that the formaldehyde fumes would be dissipated harmlessly by the volume of air passing over the cloth during the various operations. That this idea is incorrect is shown by the fact that the air in many garment sewing rooms is highly irritating.

The specification that the amount of formaldehyde released from sensitized fabric must be 2,000 p.p.m. or less has aroused great concern among the chemical producers. Since the specification went into effect much work has been done to reduce the amount of free formaldehyde in the cloth.

There are three principal methods for reducing the amount of free formaldehyde released from sensitized fabrics. The first method involves special formulation of the agent. In the manufacture of most agents, a considerable excess of formaldehyde is used to get a more nearly complete reaction. For example, the agent generally used for post-cure fabrics was made with about 2 percent excess formaldehyde in the past. Recently, there was an announcement that new formulations contain as little as 0.5 percent excess formaldehyde. The exact method of preparation of these formulations is a carefully guarded secret.

A second method is the removal of excess formaldehyde by scavenger agents in the crosslinking formulation. These agents react chemically to bind the free formaldehyde. Although these modifications are effective in reducing the amount of formaldehyde, they are expensive and sometimes they have undesirable effects on the properties of the finished fabrics (10,16).

A third approach to the problem has been directed toward removal of free formaldehyde from the sensitized fabric without costly chemical treatments or the need for elaborate equipment. SRRL researchers have found that although the formaldehyde molecule is strongly absorbed by cotton it can be replaced by a molecule of water. An inexpensive and efficient method of doing this is to expose the fabric for a few seconds to superheated steam (11). For example, cotton printcloth sensitized by treatment with a 10 percent formulation containing dimethylol methyl carbamate and zinc nitrate catalyst was passed through a chamber supplied with superheated steam (116° C.) with a dwell time of 10 seconds. The steamed fabric contained no free formaldehyde (13). The method is not expensive or complicated, and the fabric emerges dry and ready for shipment to the garment manufacturer.

Kullman and Reinhardt have proposed an extension of this steaming method (12). They show that cloth sensitized with a carbamate finishing formulation can be dried and the free formaldehyde removed in one operation by drying with steam heated to 240° to 280° F. Thus, this method makes possible the use of crosslinking agents which previously could not be used for post-cure fabric because of the excessive amount of formaldehyde present in their formulations.

Finally, two methods should be mentioned which are being used commercially to lessen the annoyance of formaldehyde in garment cutting rooms. One is a method in which the air is recirculated and cleansed. Formaldehyde vapors are not removed by the usual process of air conditioning; in fact, because of recirculation of the cooled air, the formaldehyde tends to concentrate and become stronger. However, this new method involves the circulation of air through a solution which oxidizes the formaldehyde to harmless carbon dioxide and water. One such machine rents for about 2 dollars per day and occupies only about 12 square feet of floor space. It will circulate 1,000 cubic feet of air per minute through a small scrubbing tower about 6 feet high. The only maintenance is to replace evaporated water once per day and to add plastic bags of the chemical about every 2 weeks (15). This method, of course, has no effect on residual odor in the garments after they leave the manufacturer and a similar machine is needed in the retail store which sells the garments.

Another method involves the physiological-psychological aspects of odor. In this method, a low-cost chemical is added to the treating formulation before it is put onto the cloth. The cost is only a fraction of a cent per yard. This is not a perfume intended to cover up the odor of formaldehyde. Rather, it is a material which alters what is called the "odor profile" of the formaldehyde (14).

To understand this concept, something must be known about the theory of odor. The nose is the most delicate sensory device in the human body. The odor of hydrogen sulfide, for example, can be detected in less than one part in 5 billion parts of air. Formaldehyde can be detected in an amount of about 1 part in a million (14). Some of the theories of odor have been reviewed by Freeman (2), who points out that a satisfactory theory of olfaction has defied the best efforts for explanation. However, one theory is that in some cases, an odor is detected due to the shape of the molecule as it impinges on the olfactory area. Slight changes in the shape of the molecule may entirely change or eliminate the odor of the compound. Perhaps, association of two molecules will also change the odor.

The method now being used commercially to some extent is based on this concept. A material is added to the finishing formulation which changes the odor profile of formaldehyde. That is, without this material, the nose recognizes the odor profile of formaldehyde and there is extreme psychological annoyance. When the additive changes the profile, there is no annoyance. Thus post-cure garment plant personnel can now work an 8-hour shift in places where, before the use of the additive, they refused to stay longer than 15 minutes. Their noses have been fooled into noticing a pleasant odor instead of warning the brain of a potentially serious odor.

In other words, the analytical and physiological results from the contaminated atmosphere are practically identical with or without the additive. However, the psychological results are much improved. As long as the concentration of formaldehyde in air is below the dangerous concentration, the method may be all right. Presumably, if the concentration were great, the physiological effects would be so severe as to warn the worker. However, it is also probable that workers would accept conditions with the agent which would cause them to be exposed over long periods of time to more formaldehyde than they would otherwise accept. And the hazard of long-term exposures to formaldehyde vapors is a question which is still to be settled.

SUMMARY

Nearly all crosslinking agents used in making durable press cotton garments give off obnoxious formaldehyde vapors. Thus, the removal of excess formaldehyde from treated textile products is an important problem. Although the odor of formaldehyde becomes intolerable long before a lethal concentration is reached, it is annoying and may be a health hazard to workers in the garment trade.

Much work has been done to decrease the amount of formaldehyde released from fabric because of the establishment, by one of the largest durable press garment manufacturers, of a maximum level of formaldehyde release in treated fabrics to be processed in their plants.

One method has been to reduce the amount of free formaldehyde in the formulation, sometimes by employing a scavenger agent which reacts with the excess formaldehyde used. Another method suggested is to remove formaldehyde from the sensitized cloth by treatment with steam. Futhermore, by use of superheated steam — about 116° to 138° C. —, cloth may be dried and free formaldehyde removed in a single step to produce a satisfactory sensitized fabric.

Another method of lessening formaldehyde annoyance in garment sewing rooms is the circulation of air through a solution that oxidizes the formaldehyde. Another method, which is being used commercially to a limited extent, involves the addition of an agent to the finishing solution, which changes the "odor profile" of formaldehyde. Although there is little change in the physiological action of the formaldehyde, there is a psychological effect so that the worker no longer finds the modified odor unpleasant.

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MEETING EPA REQUIREMENTS FOR REGISTRATION OF PESTICIDES

by
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ABSTRACT

Current organization of the Environmental Protection Agency and the Pesticides Regulation Division will be discussed. The requirements for registration, pending guidelines setting forth such requirements, status of pending changes in legislation, and major provisions affecting agriculture will be outlined.

DEVELOPMENT OF FOAM FORMULATIONS FROM AGRICULTURAL PRODUCTS

by
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(Presented by J.J. Spadaro)

INTRODUCTION

About 2 years ago personnel from the Agricultural Engineering Department at Louisiana State University Baton Rouge approached us at the Southern Marketing and Nutrition Research Division regarding the possibility of developing less expensive agricultural foam formulations than those being used at LSU.

The basic component in the LSU formulation at the time was edible gelation, a stabilizer which cost 90 cents a pound. This was considered uneconomical for practical application. So we took up the challenge and investigated many stabilizers, as well as foaming agents, and viscosity builders some of which were obtained from three of our sister Divisions and some from industry.

I am going to report on the development of foam formulations which contained, as their basic components, animal hide glue at a cost of 25 to 35 cents per pound or feed-grade whey solids or both at 5.5 cents per pound. Foam properties were comparable to those obtained with the gelatin formulation.

An outline of what I plan to discuss this morning is:

1. Potential uses of agricultural foams.
2. The desirable properties of foams.
3. The method of generating the foams and measuring the foam characteristics.
4. Show examples of how chemicals were evaluated.
5. Show examples of formulations for various uses.
6. Give some cost data.

Foam uses include frost or freeze protection or both, soil cover after soil fumigation; farm markers; carriers for herbicides, fungicides, and defoliants; and possibly as a cover over droppings in broiler houses to reduce odor; eliminate breeding of flies and to reduce labor costs. Persistence requirements of these foams are approximately 24 to 48 hours at or near freezing temperatures, 10 hours at 60° to 80° for soil covers, and 1 to 5 hours at 90° to 110° F. for chemical carriers and farm markers.

Desirable foam properties for frost and freeze protection include:

(1) Persistence for 24 to 48 hours.
(2) Postponement of drainage for as long as possible.
(3) Development of a "good" complete skin that would protect the foam when the wind is high.
(4) Maintaining a temperature differential between the outside temperature and that under the foam so plants under the foam would remain protected.
(5) Self-destructiveness, including skin and foam when the temperature rises.
(6) No toxicity.
(7) Ecomonic feasibility.

The above properties are applicable to other foam uses with some modification. For example, postponement of drainage is not required for the application of chemicals via foams. Figure 1 shows the laboratory model generator, which was built by the Agricultural Engineers at LSU Baton Rouge. This equipment was developed by Dr. Braud and coworkers and depends on its capacity to incorporate large quantities of air, in small bubbles, in a select liquid formulation by forcing the liquid through a series of six screens under a pressure of 35 p.s.i. Dimensions of the generator are outlined in the figure.

Figure 2 shows foam being discharged from the generator. Here you see the copious product that was obtained with one of our formulations.

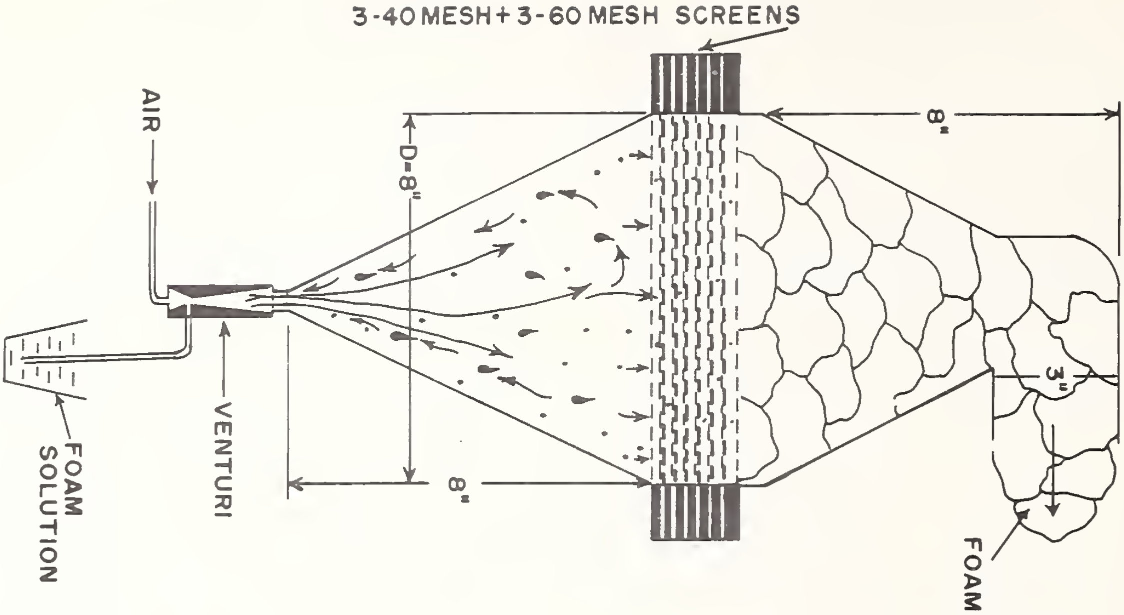

Figure 1. A cross section of the laboratory model foam generator

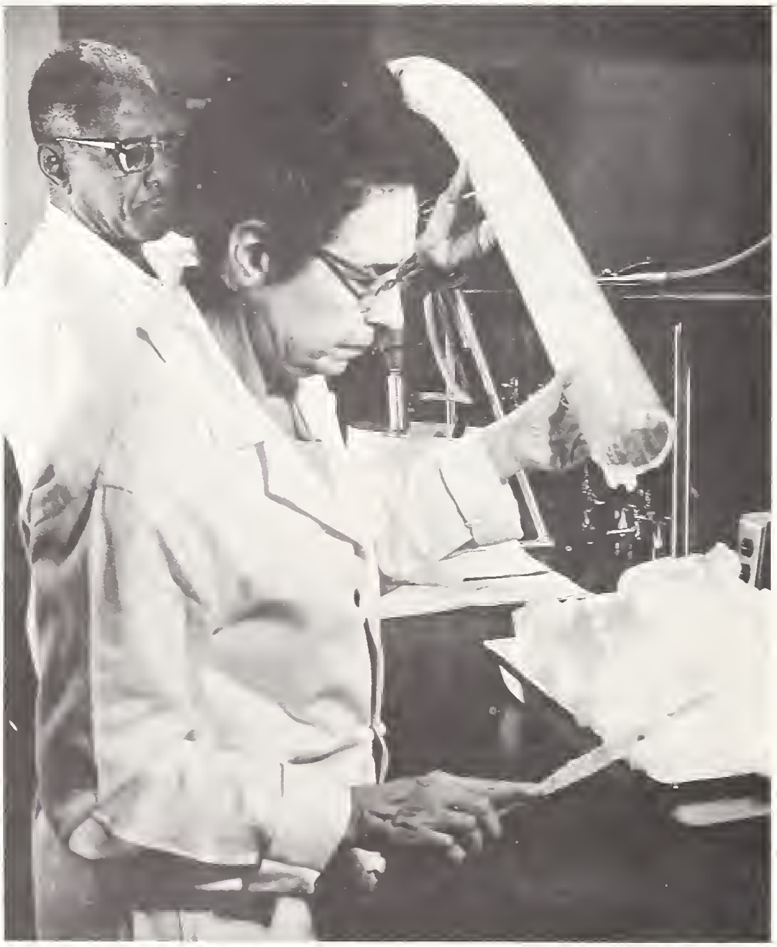

Figure 2. Foam being discharged from the generator

Metal pans (fig. 3) are filled with foam, weighed and stored at at least three temperatures, namely, 36°, 75° F., and outdoors, at from 90° to 110°. Pans seen here are at room temperature, and 100 cc. graduates have been placed under the drainage tubes to collect liquid draining from the foam. By this means we measured density in grams per cubic centimeter, expansion in cubic centimeters per gram, drainage as cubic centimeters of liquid flowing from the foam in a given time interval, and, persistence measured in hours for the foam to subside to a level of 33 percent of its original volume. Figure 4 shows a closeup of pans containing foams at critical subsidence point and skins which have been pulled aside. These techniques were used in the evaluation of all chemicals for determining their suitability for incorporation in any foam formulation.

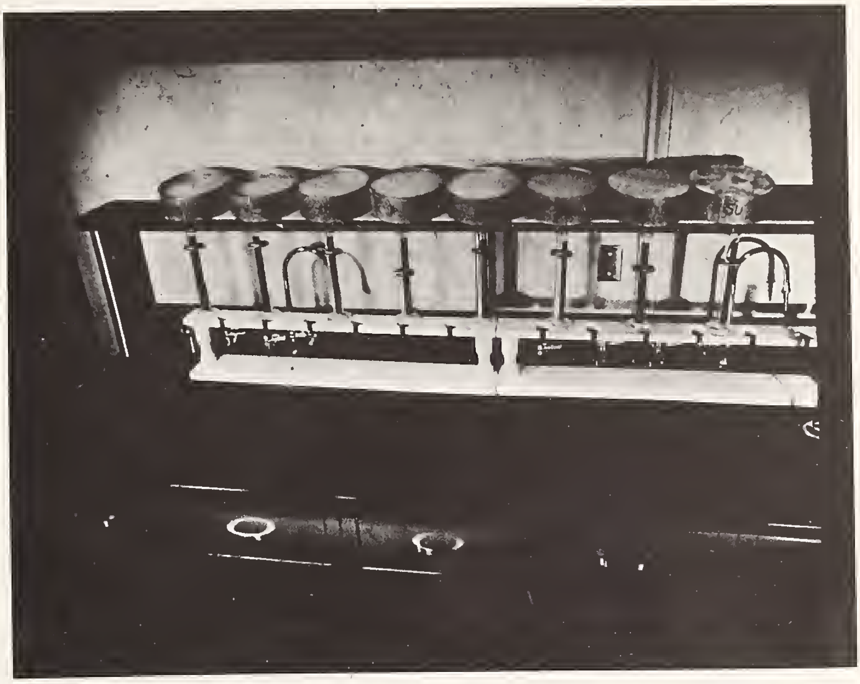

Figure 3. Metal pans filled with foams and draining into 100 ml graduates

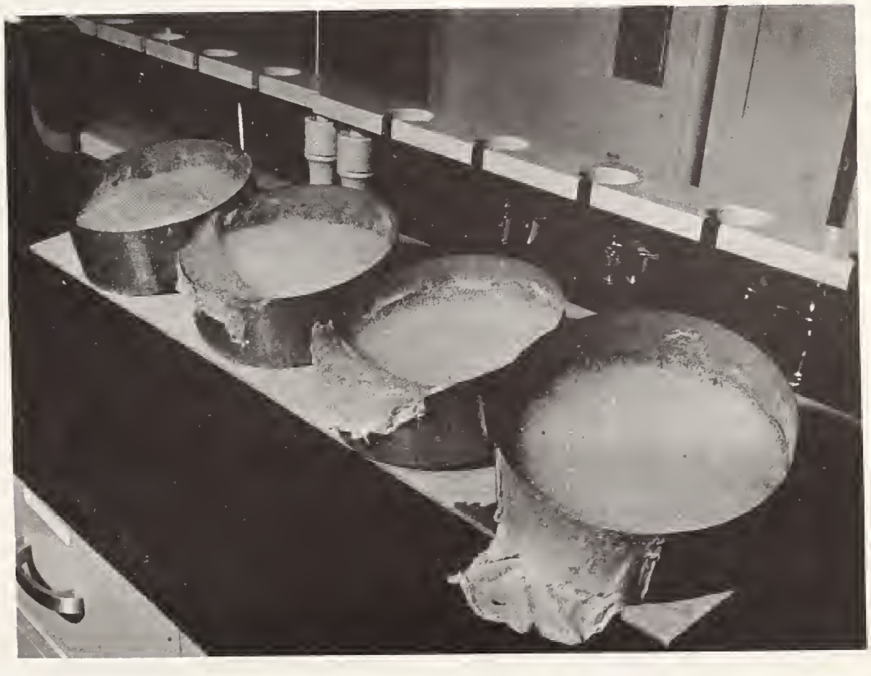

Figure 4. Closeup of pans in which foam has subsided to 33% of its original volume

Figures 5 through 8 are examples of the kinds of data obtained from these evaluations. Figure 5 shows how the selection of the surfactant, Hi-X, was made. Three other foaming agents, namely, Liquid K, Triton X-100, and Orvus K, were compared with Hi-X. Both drainage and persistence were measured and graphed as shown. Hi-X dramatically reduced drainage and enhanced persistence significantly. From this group, Hi-X was selected for the formulation.

In figure 6 the concentration of the surfactant Hi-X was varied from 0.3 to 1.9 percent. Persistence at 36° F. and density curves paralleled each other and decreased as the concentration of Hi-X increased. Greatest persistence, more than 192 hours at 36°, was obtained at 0.3 percent concentration of Hi-X where density was also highest and expansion was lowest. Extended persistence at such a low concentration of surfactant was confirmed by replicating four times. Expansion increased with an increase of concentration of Hi-X up to 1.50 percent at which point it leveled off. At 75°, persistence ranged between 7 to 10 hours peaking at 10 hours and 0.7 percent concentration of Hi-X. Persistence at 95° was poor for all concentrations.

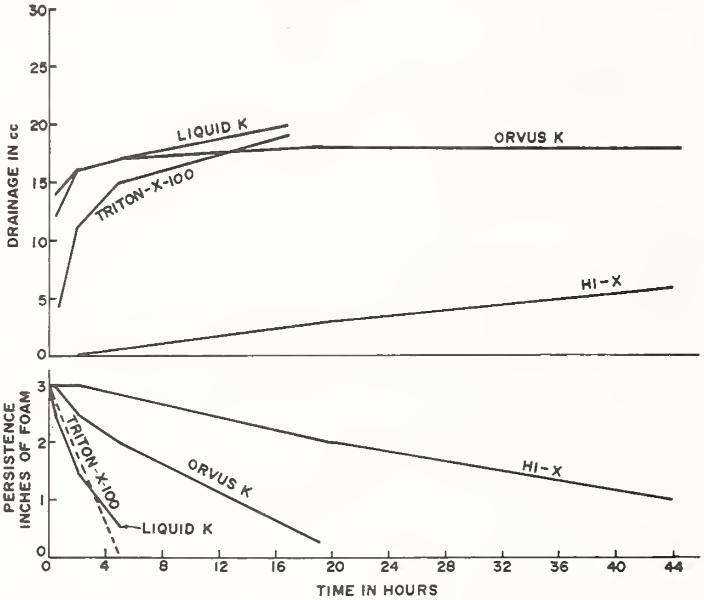

Figure 5. Drainage and persistence of foams versus time in hours for foams made with 4 different surfactants

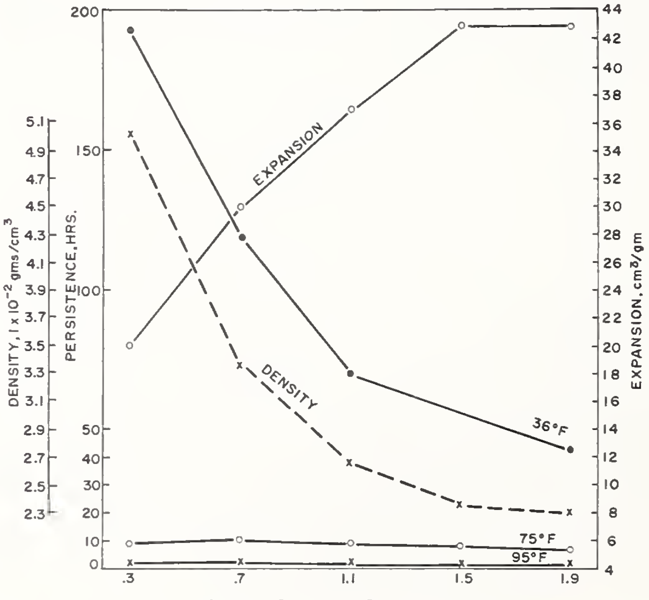

Figure 6. Effects of varying concentration of surfactant on density, persistence and expansion

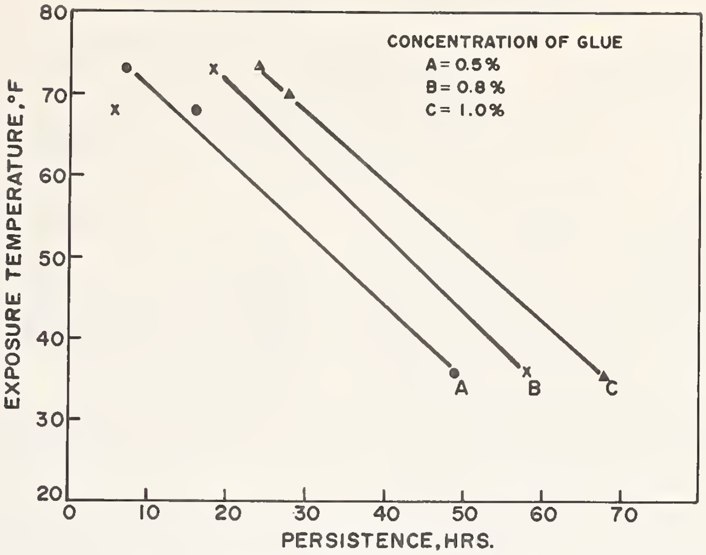

Figure 7. Effects of concentration of glue and temperature of exposure on persistence of foams

Effect of glue concentrations are shown in figure 7. In this figure, exposure temperatures are plotted against persistence in hours for three formulations, basically alike except for the concentration of animal hide glue contained therein. It was found that persistence increased with an increase in concentration of glue and a decrease in temperature.

Concentrations of the viscosity builder were also varied from 0.1 to 0.5 percent in increments of 1/10 percent. The formulation contains whey solids, animal glue, hydroxyethylcellulose (the viscosity-builder) and Hi-X. In figure 8 results are shown for both 24-hour and 48-hour formulations, that is, the formulations have been prepared 24 and 48 hours before generating the foam. As expected expansion decreased as density and the concentration of the viscosity-builder increased. Attention is called to the clustering of points of both expansion and density curves at the 0.3 percent concentration of the viscosity-builder. In that area, expansions range between 35 to 40 cubic centimeters per gram and densities between 0.025 to 0.029 grams per cubic centimeter. High expansions desirable from a cost standpoint can be obtained with lower concentrations of the viscosity-builder, such as 0.1 or 0.2 percent but very little thickening of the formulation takes place, that is, a significant viscosity-building effect is not obtained. On the basis of these data we selected a 0.3 percent concentration of hydroxyethylcellulose for specific formulations.

Figure 9 outlines examples of some formulations which we found suitable for various uses. Percent composition, expansion, and persistence at three temperature levels are outlined along with the proposed application. The first one, containing animal glue, whey solids, hydroxyethylcellulose, and Hi-X would be a good all-purpose foam judging by the expansion and persistence at the various temperatures. If, however, a foam is to be tailored specifically for application at or below freezing temperatures the next formulation would be desirable. Here you see that the concentration of animal glue, whey solids, and hydroxyethylcellulose have been increased slightly, but the concentration of Hi-X has been decreased by more than 50 percent. As a result, we lose some expansion, but increased persistence to 8 days, or 192 hours at freezing temperatures. When a foam is to be used at

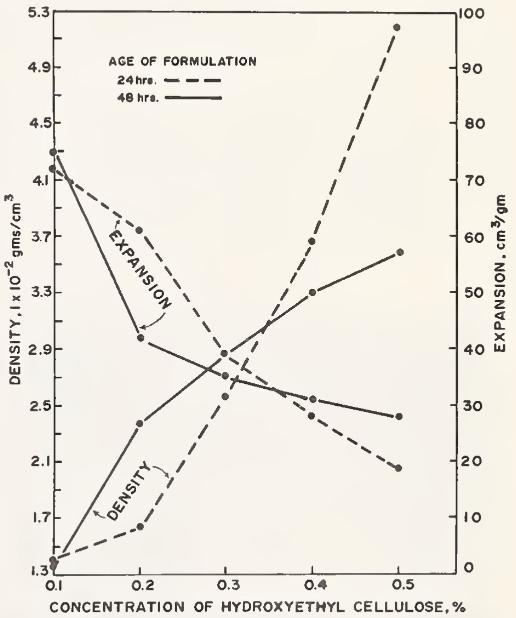

Figure 8. Effects of concentration of hydroxyethyl cellulose on densities and expansions of 24- and 48-hour formulations containing whey solids and animal glue

EXAMPLES OF FORMULATIONS

% COMPOSITION*						EXPANSION	PERSISTENCE, HRS.			APPLICATION
AG	WS	N	K	Hi-X	M-CP		36°F	75°F	OUTDOORS	
0.8	0.8	0.24		0.7		36X	135	7	3.0 (90°F)	GOOD ALL-PURPOSE FOAM
1.0	1.0	0.3		0.3		20X	192	8	1.8 (90°F)	36°F TO BELOW FREEZING
1.0	1.0		0.1		0.5	28X		14	5.3 (90°F)	90–110°F
	1.0		0.1		0.5	50X		3.5	1.5 (100°F)	SHORT-TERM FOAM
	1.5				0.5	70X	—	—	0.8	SHORT-TERM FOAM

*AG=ANIMAL HIDE GLUE; WS=WHEY SOLIDS; N=NATROSOL, HYDROXYETHYL CELLULOSE; K=KELGUM; Hi-X=SURFACTANT; M-CP=MICROFOAM–CP(SURFACTANT)

Figure 9. Compositions of selected formulations and the expansions, persistence and utilization of the foams produced therefrom

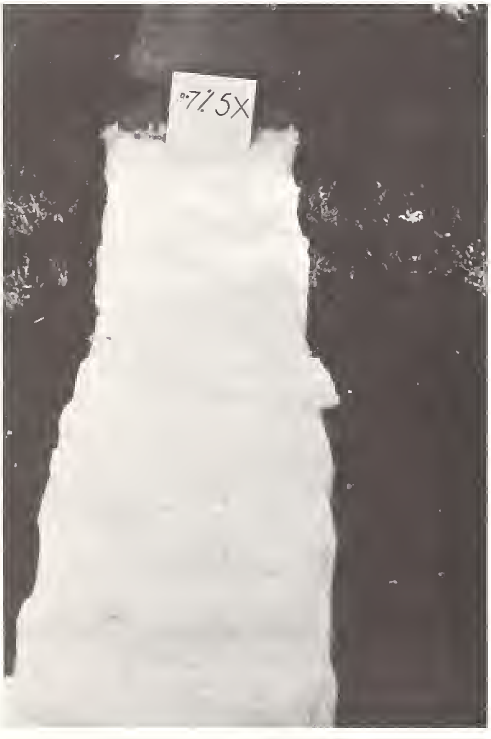

Figure 10. Blanket of foam being laid down over grass in a field

outdoor temperatures in the range of 90° to 110° F., we have found it advantageous to change to a viscosity-builder that is a polysaccharide of microbial origin (KELGUM), and to substitute Microfoam — CP (ONYX) for Hi-X. Foam from this formulation has a mediocre expansion, but has good persistence, in excess of 5 hours at 90° and 14 hours at 75°. If only a short term foam is desired we have two formulations that will be suitable. Such foams as these would make good carriers for fungicides, herbicides, and defoliants. These foams have high expansion, are relatively inexpensive, are fairly wet, and will sink down onto flowers, into whorls, and around and onto odd-shaped foliage. The whey solids contribute enough adhesiveness to the formulation to permit the foam to retain herbicides, fungicides, or other material being carried at the places where treatment is needed.

Costs of these foams range from $1.40 up to $12.70 per acre-inch for 10-inch rows and 42-inch spacings. This is considerably less than the cost of edible gelatin foams which is approximately $19.00 per acre-inch. Figure 10 shows some idea of what these foams look like when applied outdoors. This foam contains 0.7 percent whey solids, 0.7 percent animal glue, and 2 percent Hi-X.

FIELD APPLICATIONS OF FOAM

by
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ABSTRACT

The potential for foam in agriculture is discussed along with results from specific applications of foam as pesticide carrier, soil cover, and frost protection agent. Physical properties of foam are given in relation to the intended use. Mechanical equipment for foam generation in the field is discussed along with application rates, volume requirement, and costs.

FIELD APPLICATIONS OF FOAM

Foam is an interesting state of matter. It can be made with a wide range of bubble sizes and degrees of stability. There are many uses for foam in agriculture. However, only recently has widespread interest developed in exploiting the unique properties of foam. In an earlier report the author listed possible uses for foam in agriculture:

1. Carrier for herbicides
2. Frost protection agent
3. Carrier for insecticides
4. Defoliant
5. Soil cover for fumigation
6. Short life mulch
7. Carrier for cleaning agents
8. Carrier for abscision chemicals
9. Evaporation suppressant
10. Soil amendment to improve structure

This list can now be declared incomplete, since other uses for foam have been brought to our attention. Foam can be used as a ground marker for field operations. Bintner (5) reported the development of foam nozzles for aerial application of pesticides. The main advantage claimed for foam-spray is that drift of fine particles is greatly reduced, allowing the pesticide to hit target and not contaminate adjacent areas. Application can be made in windy conditions which would prohibit spray application. The visible evidence of foam-spray eliminates the need for a flagman since the pilot can see exactly where material is applied.

Byrom (9) used foam to apply sucker-control chemicals to tobacco plants. Here, foam was utilized to carry the chemical to its intended target in the plant structure. A similar approach for corn borer control was reported by Berry (1), Agricultural Research (1971).

The formulation and method of generation of foam must be designed according to its end use. Definition of certain physical properties of foam is necessary to describe its nature. Foams are inherently unstable systems. Freshly generated foam will immediately undergo physical changes unless it is stabilized, so physical properties can be time-dependent. To the user of foam the main properties of interest are expansion, drainage rate, life (or persistence), thermal properties, and end residue.

Expansion is the reciprocal of foam density in metric units and the reciprocal of specific gravity in English units. Volumetric expansion is often expressed in units gallons of foam/gallon of solution. The density of foam is easily measured by catching a tared container full and measuring the weight of the known volume.

A method of characterizing foam drainage is based on time required for one-fourth of the liquid volume to drain. The quarter drainage time is measured by placing a pan of freshly generated foam on a slightly tilted shelf and collecting the liquid drainage. Evaporation of liquid from bubble walls causes loss of foam weight. Its magnitude would be significant in a windy, dry atmosphere. For foam exposed to environmental conditions excellent for evaporation, foam loss due to mass loss might be more destructive than weakening due to drainage. Drainage causes a loss of liquid and is accomplished by bursting of some bubbles, but the loss of bubble structure does not necessarily occur as fast as drainage.

Persistence is the property of primary interest and least reported in the literature. Foam persistence is the measure of foam volume retention with time. There seems to be no standardized method for measuring it. The decrease in thickness of a foam layer with time can be used as a measure, or the time required for half of the volume to disappear, a half life concept.

Persistence values are meaningful only when the physical conditions of the environment causing the destruction are given. Major elements of destruction are wind forces in the field, and dust in the atmosphere.

FOAM GENERATION

The mechanical function of a foam generator is essentially to mix air with the foam solution in such a manner that large numbers of small bubbles are produced. Foam generation is a dynamic process in which all factors must be "right" for foam production. To produce homogeneous foam, the air-liquid ratio must be adjusted to uniformly mix into the liquid just the amount of air the solution is capable of capturing in bubble formation. The quantity that can be captured depends on the chemical properties of the foam solution. Energy must be imparted to the mix to overcome surface-tension forces, either by mechanical and/or hydraulic agitation. Energy requirements are a function of the efficiency of the mixing process and the richness of the lather. Energy requirement is inversely related to the bubble size produced. Theoretical aspects of energy considerations are given by Bikerman (2,3). Details of design of foam generators for laboratory use and for field application of foam are given by Braud (7,8). A field model is shown in figure 1. A foam pump is now being sold by the Waukesha Foundry Company, Inc., Waukesha, Wis., (figure 2).

FOAM FOR FROST PROTECTION

The insulating ability of a blanket of foam makes it an excellent medium for protecting low growing plants from freezing, Simonovitch and others. (11) Foam for frost protection must be stabilized to last overnight at least. Expansion values of 35 to 100 have been used.

A 4-inch deep foam insulation blanket proved more than adequate to prevent freeze damage to strawberry plants, Chesness (1968). Although the mimimum recorded exposed surface temperature was 21° F., temperatures within the 2-inch foam layer adjacent to the ground surface never reached freezing in any of the five tests.

The foam generated from a 2 percent foaming agent (Orvus K) plus 2 percent gelatin (7 APM) mixture with expansion 46 exhibited the highest degree of persistence, 62 hours. This foam provided two consecutive nights of freeze protection with minimum exposed surface temperatures of 25° F. Despite this herculean performance, the tough skinlike residue from this mix and high cost ($200 per acre) precludes our recommending it for field use. Low-cost stabilizers formulated by Madeline Lambou and J. Spadaro have been laboratory tested and given preliminary field tests. The cost of band application for frost protection can possibly be reduced to less than $100/acre. One commercial mix "Agri-foam" (Laurentian Concentrate Company, Ottawa, Ontario, Canada) is now being marketed in several specialty crop areas.

FOAM COVER FOR SOIL FUNIGATION

Soil fumigation is a widespread practice in the production of many crops. A toxic gas is

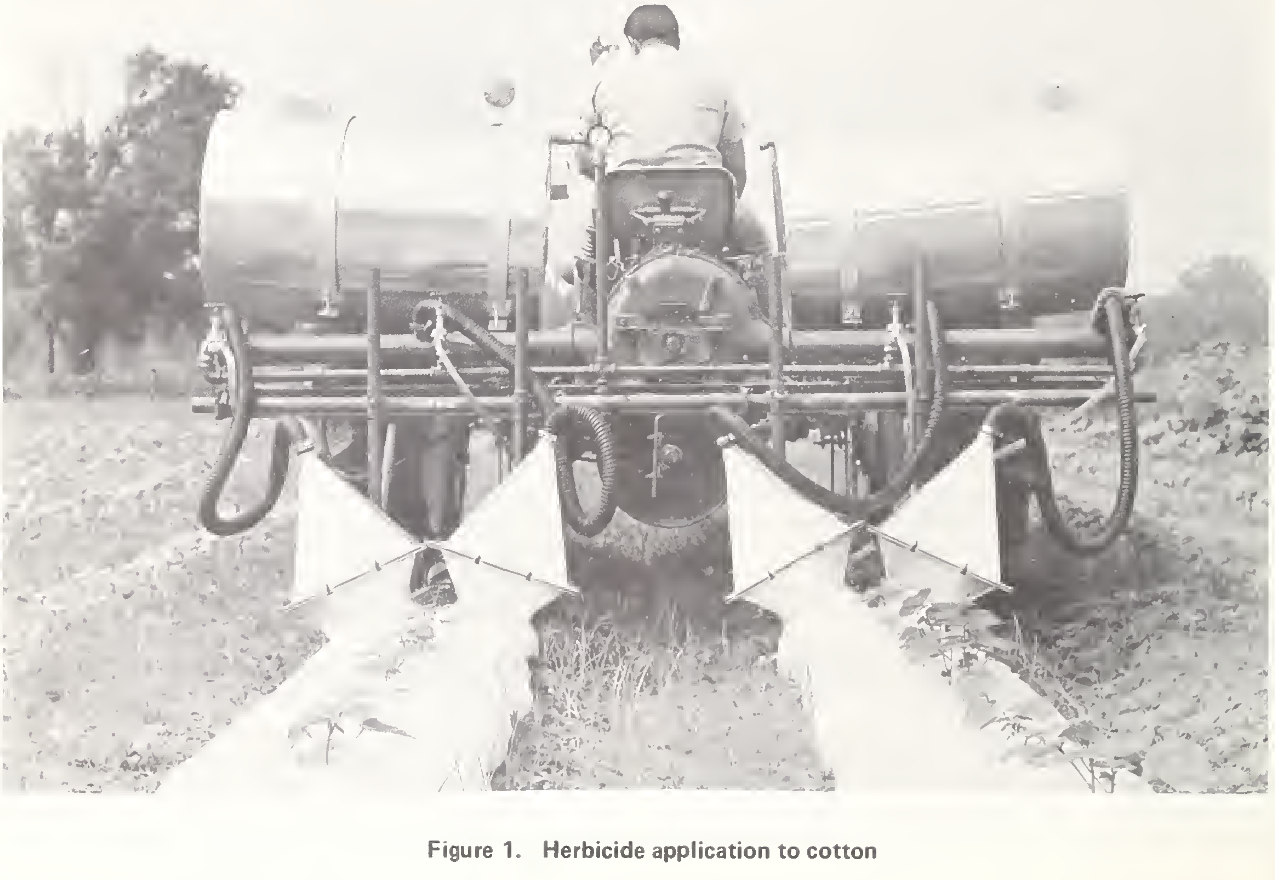

Figure 1. Herbicide application to cotton

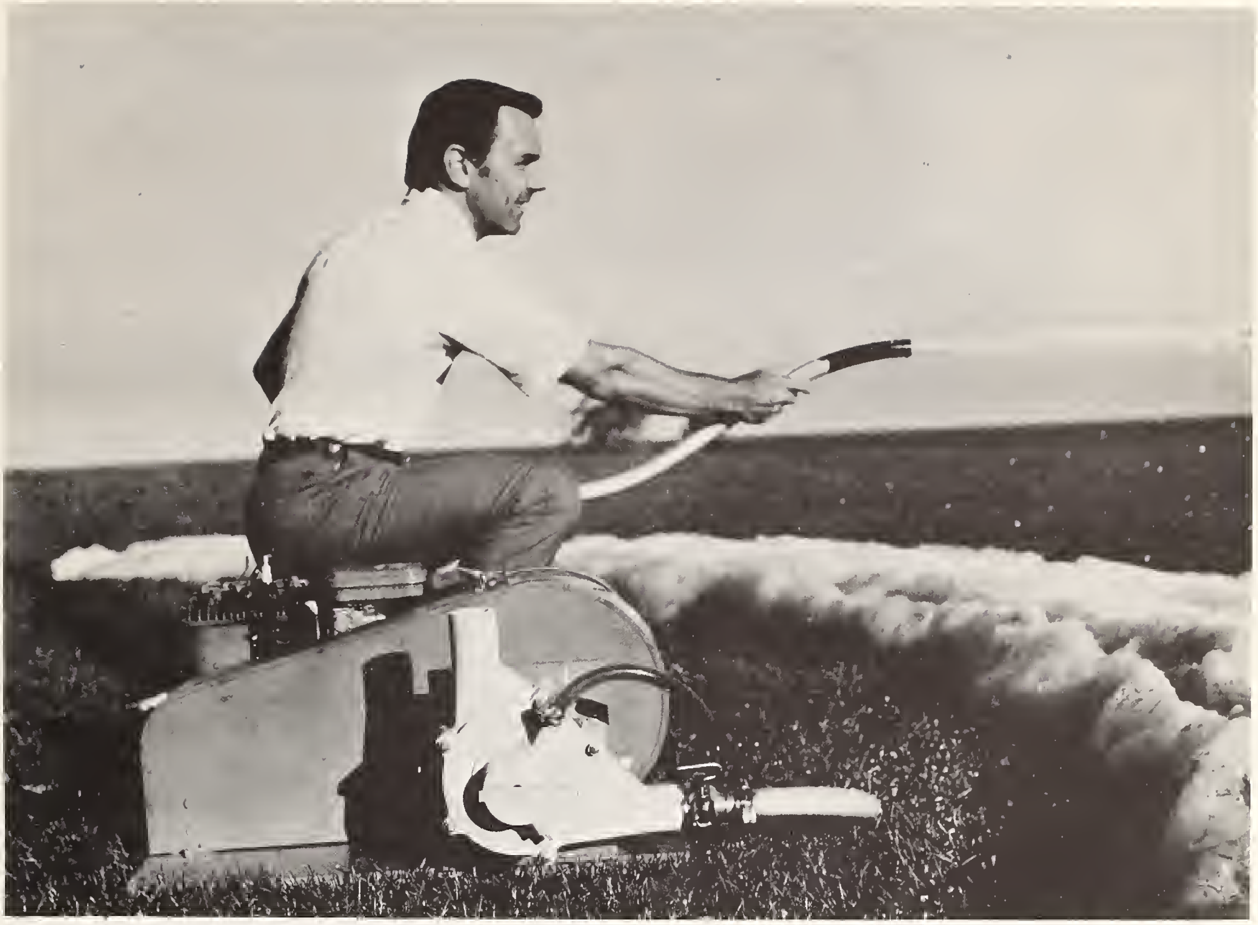

Figure 2. Waukesha foam pump

used to fumigate the entire soil root zone in tree seedling nurseries, greenhouses, and in fields for tomatoes, tobacco, and other crops.

Formulations of volatile, toxic gases are employed for fumigation purposes; especially mixtures of methyl bromide, ethylene dibromide, and related compounds. These formulations are applied either as a pressured gas under a soil cover or in a gel carrier which is incorporated into the soil and releases the gas slowly. The gel form of fumigant will give partial soil treatment without a soil cover; however, a cover placed over the soil provides better treatment.

Several methods of sealing the soil surface have been tried in the past. In 1969 and 1970 field studies in the use of a foam layer for sealing soil fumigated with gelled fumigant were conducted in cooperation with two commercial firms who do custom soil fumigation. A 4-inch layer of foam placed over soil plots fumigated with gelled methyl bromide increased effectiveness of soil treatment. Improvement in weed and nematode control resulted with foam. Cost was comparable to film cover. Twenty-five percent reduction in fumigant rates gave as good results as standard rates. See tables 1 and 2.

Table 1. Weed count

Days after treatment	Methyl bromide 20 g.p.a.	Methyl bromide 20 g.p.a. + foam	Methyl bromide 15 g.p.a.	Methyl bromide 15 g.p.a. + foam	Foam only	Check
			Weeds/ft.^{2}			
12	30	14	29	3	30	25
19	53	7	23	5	47	63
27	31	3	22	3	28	28

Table 2. Nematode count

Soil treatment	Nematodes per pint of soil
Methyl bromide 20 g.p.a.	400
Methyl bromide 20 g.p.a. + foam	100
Methyl bromide 15 g.p.a.	420
Methyl bromide 15 g.p.a. + foam	146
Foam only	1,286
Check	2,705

Volume and cost per acre

For foam applied on bands 4 feet wide, 4 inches deep with 6 feet spacing between rows, the volume of foam required per acre is as follows:

$$V = 302.5 \frac{w.d}{S}$$

$$= 302.5 \frac{(4 \times 12) . (4)}{6} = 9{,}680 \text{ ft}^3$$

where w = band width, inches
d = foam depth, inches
S = row spacing, feet
V = foam volume, ft^3

Cost of foam actually used: 2 percent starch phosphate + 2 percent Jet-X
Volume of water required = 1810.0 gallons per acre
Expansion = 40
Cost per pound of starch = $0.17
Cost per pound of Jet-X = $0.75
Cost per acre = $260.00

The high cost ingredient here is Jet-X, a specially formulated foaming agent for fire fighting use. Some synthetic detergents with excellent foaming ability cost $0.20 per pound.

Total costs for fumigant plus foam application in this study were: For 20 g.p.a. fumigant at $15 per gallon + $260 = $560 per acre, and for 15 g.p.a. fumigant at $15 per gallon + $260 foam costs = $485, which is comparable to costs with film cover.

In the soil fumigation tests, reduction in fumigation rates was possible with foam cover. Good nematode and weed controls were obtainedwith 25-percent reduction in fumigant rates. Since gelled methyl bromide costs about $15 per gallon. at 20 g.p.a., a 5-gallon saving is worth $75 per acre.

Research and development in the formulation of low cost foam is underway at the Southern Marketing and Nutrition Research Division Laboratory, ARS, USDA, New Orleans, La. Stable foams have been laboratory- and field-tested (not with soil fumigation) that would lower foam costs to about $75 to $100 per acre. If higher foam expansion can be developed — and we have good reason to believe it can — even lower cost would result. Our goal is to reduce foam costs to about $50 per acre.

FOAM—HERBICIDES STUDY[1]

To evaluate possible benefit of using a foam to improve the efficiency of herbicides, two greenhouse studies were made by using foam as a carrier with surface applied herbicides. The experimenters hope that less herbicide material would be lost to the atmosphere immediately following treatment, less surface soil moisture would be lost, a higher soil temperature would be reached, and the conditions would be improved for the activity of the herbicide.

Seeds of several weeds (barnyardgrass, sida, Johnsongrass and morningglory) were planted in pans. One percent surfactant was used as the foaming agent. All applications with or without foam were made under 500 ft. per min. (5.7 m.p.h.) wind velocity. In foam treatments, the herbicide was added to the foam solution and applied as a shortlived foam. Foam life was 5 to 7 minutes. In nonfoam treatments, the herbicide was applied with a conventional flat fan nozzle under 30 p.s.i. pressure.

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[1] Credits: M. Esphahani, W. J. Blackmon, S. H. Crawford

Pans treated with foam showed signs of injury to the weeds one day after treatment. Foam caused minor injury to cotton and severe injury to soybean plants. One week later, weeds in all pans which received herbicides in form of foam or spray were completely killed. Table 3 gives the weight of the remaining plant materials.

Table 3. Herbicide application in foam

Herbicide	Rate	Method	Weeds	Soybeans	Cotton
			G./sq.ft.	G./sq.ft.	G./sq.ft.
Cotoran	2 lb. 80 pct. weeds per acre	Foam	0	3.1	0
Do	1 lb. 80 pct. weeds per acre	do	0	5.5	0
Do	2 lb. 80 pct. weeds per acre	Spray	0	0.5	2
Do	1 lb. 80 pct. weeds per acre	do	0	1	1
Tenoran	3 lb. 50 pct. weeds per acre	Foam	0	0	0
Do	1.5 lb. 50 pct. weeds per acre	do	0	1.8	0
Do	3 lb. 50 pct. weeds per acre	Spray	0	2	3.0
Do	1.5 lb. 50 pct. weeds per acre	do	0	5.8	10
Check	-----	---	26	5.0	11
Foam only	-----	---	26.5	4.5	0

The amount of herbicides at both levels was too high, since weeds in all treatments were completely killed. It was decided to repeat the test with a reduced herbicide level.

In the second test, pans treated with foamed herbicide again showed signs of injury 1 day after treatment. Cotyledons of cotton plants suffered minor injury, but soybeans could not tolerate the combination of foam and herbicide — they were either killed or heavily injured. Weeds in plots with foam-herbicide at all levels showed a good degree of control. Table 4 gives the weight of plants per square foot of plot.

Conclusions

At the levels of concentration of both herbicides used, there is a definite difference in the toxicity of the foam applications in comparison with the herbicide applied as a topical spray, the foam application being much more toxic. These differences are of such magnitude that at the lowest level of herbicide concentration in foam, almost 100 percent weed control was obtained in the foam application as opposed to essentially no control with the topical spray.

Broadcast application of short-lived foam with herbicide appeared to be possible in cotton since good weed control was obtained with negligible injury to the cotton. Injury to soybeans was too great to permit direct application of either material in a foam. Care must be taken that foam does not come in contact with soybean plants.

One possible reason for apparent increased activity of the foamed plot was the elimination of drift conducted under condition of 500 ft. per min. wind velocity. There was loss of fine spray particles while the foam was apparently unaffected.

Herbicide application, field study

Foam for postemergence herbicide applica-

Table 4. Herbicide application in foam

Herbicide	Rate	Method	Weeds	Soybeans	Cotton
		G./sq.ft.	G./sq.ft.	G./sq.ft.	G./sq.ft.
Cotoran	1 lb./acre	Foam	0.9	0	1.95
Do	1/2 lb./acre	do	2.5	0	6.4
Do	1 lb./acre	Spray	20.8	2	2.25
Do	1/2 lb./acre	do	16.5	3	5.1
Tenoran	2 lb./acre	Foam	1.4	0	5.5
Do	1 lb/acre	do	0	0	3.4
Do	2 lb./acre	Spray	38	3.5	1.65
Do	1 lb./acre	do	15.5	4.0	4.8
Check	-----	---	45.5	3.0	3.9
Foam only	-----	---	23.3	0	1.5

tion on cotton was compared with conventional spray application in field tests in 1971. Field plots on Mhoon Silt Loam soil were planted to cotton in late April. Dense week growth was allowed to develop before application of postemergence treatments. Three herbicides at two application rates, were used with four types of application: (1) Conventional spray, flat fan pattern; (2) foam nozzle; (3) heavy foam carrier; and (4) lightweight foam carrier. All treatment combinations were replicated four times.

Application methods

Conventional Spray — Spraying Systems Company No. 8001 nozzles at 30 p.s.i. directed spray. Foam Nozzle — Delavan Manufacturing Company, "Dela-foam" No. 9 and 4 as wet foam directed in fan pattern. Heavy Foam — LSU foam generator utilizing air flow and brass screens to create foam from premix to 2 percent Hi-Ex fire foam solutions plus 2 percent corn starch phosphate. This mix produces foam with expansion of 35 volume per volume. Herbicide was added to foam solution. Light Foam — LSU foam generator utilizing herbicide solution directly. All herbicide treatments contain 1 percent surfactant (Ortho X-77). This allowed production of copious foam with the foam generator. Foam expansion was about 250 volume per volume.

Treatments with conventional spray and heavy foam were applied in 16-inch wide band broadcast over weeds and cotton in row. Foam band thickness was about 4 inches. With heavy foam, life average 3 to 3½ hours. Damage to weeds and small cotton plants covered with heavy foam was noticed as soon as foam was dissipated. It appeared that foam nozzle did not give a uniform distribution of the liquid over the plot area. Application of light foam was very successful in 10 to 15 m.p.h. wind. Rate of foam generation was fast and, surprisingly, high wind velocity did not remove the foam from the covered area. Foam had the expected life of less than 5 minutes, allowing drainage of herbicide solution down into the plant canopy.

Findings

The results of the experiment, table 5, particularly in treatments with light foam for carrier for herbicide, were very encouraging. Since this test was conducted at a late stage of growth of the weeds, one could expect to obtain even better weed control when weeds are at proper age for postemergence herbicide application. Light foam intensified the effect of herbicide on weeds to a large extent. Light foam is easy to produce, would cost $1 to $2 per acre, and gave better weed control than heavy foam which would cost much more for stabilizers. The foam nozzle was the same in action as conventional spray. Since light foam uses surfactant already present in the spray solution for foaming, the cost of foam is only about $1 per acre added. Further work is needed to fully explore reduced herbicide rates in foam.

One disadvantage for the foam application method is a greater water requirement than is used with spray application. In the above study, about 80 gallons per acre was required for the foam application. A lesser volume of foam probably could be used. McWhorter and Barrentine (10) used foam application with 40 gallons of water per acre. Further study is needed to learn what effect foam volume has on herbicide efficiency.

Second Test

This experiment was conducted at the time that weeds were at proper stage of growth for postemergence treatment. The purpose was to test effectiveness of herbicide (table 6). Karmex

Table 5. Weed control in cotton with different methods of herbicide application, Louisiana Agricultural Experiment Station, Essen Lane Farm, Baton Rouge, La.

Herbicide[1]	Rate	Method	Rating of weed kill[2]	Rating damage to cotton
	Lb. per acre			
Karmex	0.5	Foam nozzle	1.3	0.5
do	.5	Light foam	9	0
do	.5	Heavy foam	8.3	4.8
do	.5	Spray	1.8	0
do	.25	Foam nozzle	1.3	1
do	.25	Light foam	7.5	0
do	.25	Heavy foam	7.3	2.5
do	.25	Spray	.75	0
Cotoran	1.0	Foam nozzle	1.5	1.3
do	1.0	Light foam	7.9	0
do	1.0	Heavy foam	7.3	6.5
do	1.0	Spray	.5	0
do	.5	Foam nozzle	.75	.5
do	.5	Light foam	4.75	0
do	.5	Heavy foam	6.3	4.3
do	.5	Spray	1.5	0
Caparol	.75	Foam nozzle	1.8	1
do	.75	Light foam	7.5	.25
do	.75	Spray	1	.5
do	.375	Foam nozzle	1	1
do	.375	Light foam	4.5	0
do	.375	Spray	1	0
		Foam nozzle only	.5	0
		Light foam only	.25	0
		Heavy foam only	5.0	3.5
		Check	0	0

[1] Date of treatment: May 25, 1971. Date of rating: June 2, 1971.

[2] The rating in the table is 0 to 10; 0 for no kill and 10 for perfect kill. Each value is the average of four replications.

Table 6. Weed control in cotton with different methods of herbicide application[1], Louisiana Agricultural Experiment Station, Essen Lane Farm, Baton Rouge, La.

Herbicide	Rate	Method[2]	Annual grass	Bracheria	Pigweed	Morning-glory	Sesbania
	Lb. per acre						
Karmex	0.5	Foam	7.5	7	8.7	8	8.7
Cotoran	1.0	do	2	3.2	4	3.2	2
Caparol	0.75	do	8.7	8	8.7	8.2	8.7
MSMA	2.0	do	3.7	2.5	0.8	2.2	2.5
Herban	1.0	do	[3]	[3]	[3]	[3]	[3]
Caparol	1	Spray	5.7	5.2	8.5	7	7.5

[1] Each value is an average of four applications.

[2] Light foam (1 percent X-77 Ortho) was used in all treatments. The rating was done from 0 to 10. Zero for no kill and 10 for perfect kill.

[3] No control.

and Caparol with light foam gave a good degree of weed control. Only Caparol spray and foam were directly comparable, and Caparol with foam gave better weed control than the spray. In this test both the weeds and the cotton plants were covered with foam.

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SESSION IV: "Consumerism" — Ray C. Frodey, Gerber Products Company, Fremont, Mich., Chairman

HEALTH AND SAFETY ASPECTS OF NEW FOOD PRODUCTS

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The dry food processing industry, as distinguished from the canning, freezing, or dairy processing industries, prior to 10 years ago concerned itself only with areas of macro contamination, more for esthetic reasons than because of the health hazards involved. With widespread use of agricultural chemicals and the development of gas chromatography which permits determination of parts per billion, the complexity of product safety mushroomed into a massive technological undertaking.

Micro-contaminates and naturally occurring toxicants have made it necessary for the food industry to increase its technological capability in all aspects of production — design of a product, specifications for the raw materials, processing and packaging conditions, storage and warehouse facilities. Even potential misuse of a product by the ultimate consumer might lead to a public health hazard. The mechanisms for implementing these safety checks are intricate and require painstaking attention to detail. Their purpose is twofold, to protect the consumer and to protect the corporation and its officers.

To accomplish these objectives, it is necessary that applicable laws be taken into consideration. The Food, Drug, and Cosmetic Act of 1968 contains the following provisions:

> A food shall be deemed to be adulterated if it bears or contains any poisonous or deleterious substance which may render it injurious to health.
>
> . . . if it consists in whole or in part of any filthy, putrid, or decomposed substance
>
> . . . or if it has been prepared, packed, or held under insanitary conditions whereby it may have become contaminated with filth.

The definition of a hazardous substance, according to the Hazardous Substances Act of 1969, is:

> Any substance or mixture of substances

which (i) is toxic, (ii) is corrosive, (iii) is an irritant, (iv) is a strong sensitizer, (v) is flammable or combustible, or (vi) generates pressure through decomposition, heat or other means, if such substance or mixture of substances may cause substantial personal injury or substantial illness during or as a proximate result of any customary or reasonably forseeable handling or use, including reasonably foreseeable ingestion by children. A public health hazard, accordingly, might be one that could result in death or injury to a consumer, whereas a regulatory hazard for example, could be salmonellae in chocolate or a DDT residue.

PRODUCT SAFETY

Product safety requires safeguards to protect against results of hazards. Illness might result from microbiological contamination of raw material (salmonellosis), improper processing (botulism), personal contamination (staph), or infected animals (clostridium). Illness might also be caused by chemical poisoning (pesticides, paints, or accidental contamination during shipment); it might be psychosomatic, if for instance, the consumer found a repugnant material in the processed food.

Other safeguards are needed to protect the consumer against injury that might result from processed foods, such as glass or metal slivers becoming imbedded in the alimentary canal, broken teeth from coming unexpectedly in contact with hard objects, or explosion of cans.

The food processor must protect the product against macro contaminants from insects, rodents, birds, glass, metal, hair and a wide variety of substances such as wood splinters, sugar balls, and plastic contamination from carriers or containers. Micro-contaminants include bacteria; yeasts; molds; viruses; pesticide residues such as rodenticides and herbicides; heavy metals; naturally occurring toxicants such as mycotoxins and solonine; and contamination during shipment.

Additionally, food additives could constitute hazards. Some of the additives are intentional for protection or enhancement of the product, such as nitrites, sulfites, antioxidants, bleaching agents, food colors, or flavorings. Incidental additives include greases and oils, boiler water compounds, plastic release agents (phythalic esters), contamination of packaging material and biphenyls polychlorinated.

MECHANISMS OF CONTROLLING

In controlling product safety, the initial step is to design a product that will of itself eliminate as many hazards as possible. This involves choosing approved raw materials, including additives that are efficacious as well as safe, and critical ingredients. The next step is devising a suitable formula. Conditions the product may encounter in use must be considered, such as shipping and storage limitations, possibility of misuse, and communicating to the consumer the proper information needed for storage, preparation, and so forth.

Processing of the product must take into account equipment design, environment, and the importance of minimizing the human element that invariably enters into operations. Finally, packaging must be concerned with ease and safety of shipping, code dating, and protection of the product against factors such as moisture, infestation, and physical damage.

RAW MATERIALS

The purchase of supplies used in a food product are of great importance. All suppliers of critical ingredients are inspected before approval is given to purchase. Suppliers are instructed to advise if there is any FDA interest in their products. Specifications must be issued on all ingredients before purchasing. All suppliers must provide a statement that their products meet FDA standards. Every shipment is inspected and tested when appropriate on arrival in the plant, that is, corn is visually inspected, albumen samples tested for Salmonella, and malt flour is tested for aflatoxins.

PROCESSING

The primary responsibility for cleanliness and sanitation rests with production. Most plants have a plant sanitarian. Inspections of the plant are made at regular intervals; a quality control audit is made monthly, and a corporate quality control report made semiannually. Random environmental samplings are made for microbiological contamination. These are inhouse measures; additionally, the plant is inspected annually by representatives of an outside inspection service and by various regulatory agencies. All plants must notify the Director of Quality Control and Nutrition of an FDA inspection and make a written report within 24 hours following the inspection. Appropriate followup action is taken and confirmed on all inspections. Any critical situations are reported to the president of the company.

Food and Drug Administration has issued guidelines on Good Manufacturing Practices, including desirable structural and equipment needs. They recommend that good personal hygiene be stressed, such as medical examinations on hiring an employee and specific policies to be observed on the job.

The choice and use of pesticides, sanitizers, and lubricants in the plant must have corporate approval as an added precaution against use of a substance that might prove hazardous.

FINISHED PRODUCTS

Packaging materials and operations must be tested and approved, including canning procedures, storage instructions, and packages that are tamper-proof. Boxcars and other carriers are inspected to be certain that physical conditions and handling of the product are satisfactory. All warehouses, where title to the product stored is held, are inspected annually; all products are code-dated to guard against sale of a product that may have deteriorated through aging.

In instances where the product is produced and packaged under contract, rather than under the direct supervision and control of the corporation, all potential operators must be inspected and approved before the contract is signed. Their technical capability is assessed to determine if they can provide adequate protection. If this is satisfactory, an audit program is established involving reports and out-turn samples; if not, a corporate quality control man is assigned during production runs to assure product quality and safety while assisting the contractor to develop his own capability.

The food processing plant is in a sense the kitchen of millions of consumers. The corporation has a moral obligation to assure its customers of purity, safety, and wholesomeness in the products purchased from it. Product safety is the process of constantly adjusting to new technical knowledge and educating employees in the use of that knowledge. The Director of Quality Control must be constantly alert to enforcement of new and tighter standards to constantly and consistently improve food supplies.

NUTRITIVE PROPERTIES OF PROCESSED FOODS

by
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A very large fraction of our diet today is made up of processed foods. In fact, if you will accept a very broad definition of the term "processed foods," it is accurate to say that virtually all of the foods consumed by our largely urban population have undergone some form of commercial handling. The "fresh produce" that we buy in a supermarket has been subjected to a commercial washing and packaging operation, and is frequently transported long distances from its point of production. "Fresh meats" may have originated in large scale feed lot operations where the cattle are raised in assembly line fashion on commercial feed formulations. Has a "hot house" tomato been commercially processed? How about the "green wrapped" tomatoes that we use much throughout the winter?

You may be unwilling to include some of these extreme examples under the heading of "processed foods." The fact remains that virtually all of our food supply has been subjected to some kind of preparation by a commercial concern. This is all very convenient and few consumers would be willing to sacrifice this convenience if they had a choice.

Nevertheless, these food preparative operations are carried on outside of the observation and control of the consumer. In a society that, not so very long ago, was almost totally agrarian, it seems to some observers to be "unnatural." Some are left with a feeling of uneasiness. What is this total dependence upon massed produced foods doing to us? Are new production methods changing the composition of foods in any important respect? Are subtle changes induced by mass production or commercial processing methods depriving us, somehow, of essential nutrients?

Food preservation by canning is one of the oldest commercial food processing procedures still in widespread use. Perhaps a review of its nutritional programs will help us to view these questions in clearer perspective.

Commercial canning operations in the United States began 150 years ago, following an even earlier beginning in England and France. This original "convenience food" was the object of similar questioning early in its history. For example, knowledge about vitamins began to accumulate shortly after World War I. Naturally there were questions about the effect of canning operations on these mysterious substances recently demonstrated to be essential to life. In 1922 a staff member at the National Canners Association Research Laboratory reviewed the literature and published "Vitamins in Canned Foods," the first technical publication on nutrition to be issued by the NCA Research Laboratory (Kohman, 1922)[1]. The literature evidence assembled by E. F. Kohman provided good reason to believe that the vitamins then recognized were conserved in useful quantities by commercial canning methods.

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[1] Kohman, E. F. Vitamins in canned foods. National Canners Association Bull. 19-L 1922.

Experimental work was begun immediately under a cooperative arrangement with Columbia University, with Kohman in charge of the canning technology and W. H. Eddy directing the nutritional assays. This collaboration continued for 15 years and resulted in 17 papers published from 1924 through 1938. The work did much to establish canned foods as nutritionally sound products and to dispell many of the questions about possible loss of vitamins during commercial processing. A further development of this early work was the first extensive vitamin C survey of the tomato juice industry carried out by Sanborn[2], an associate of Kohman, and published in 1938. This work defined many of the plant operating conditions required for the conservation of vitamin C.

Of course, much of this early work was very qualitative in nature. The only available analytical procedures were extremely cumbersome and time consuming. During the 1930's, knowledge of nutrition was increasing rapidly and the tools for making it more quantitative were becoming increasingly available.

In 1940 nutrition became a factor in the Nation's defense. The operation of the selective service system afforded the first opportunity in many years to evaluate the nutritional status of the young men of the country. During the first few months of operation, it became apparent from the physical examinations that approximately one-third of the men called for induction showed definite evidence of malnutrition and many were rejected on this account. This fact and the necessity for developing adequate army rations resulted in the calling of the National Nutrition Conference for Defense, in Washington in May 1941. This conference pointed out the need for more complete quantitative information on the nutritive properties of all types of foods to permit better planning and improvement of military and civilian diets. The canning industry responded to this need with a major research effort designed to collect definitive information on the nutrient content of canned foods and to develop the information necessary to insure maximum nutrient retention in commercial processing operations.

This major research effort was carried out in the laboratories of the National Canners Association and cooperating can manufacturers, and, by means of research grants, in a large number of cooperating universities throughout the United States. Active research was in progress in 1942, and the program was continued over a period of 12 years by workers in 14 laboratories. The results were published in a series of 45 original scientific contributions. There were numerous review articles, including a widely distributed test published in 1950, "Canned Foods in Human Nutrition." This work still provides the foundation for much of the available scientific information on the nutritive properties of canned foods, and the factors affecting nutrient retention during commercial processing.

During the 1950's a major nutrition effort was carried out in the frozen food industry to define the nutritive content and the optimum conditions for storage and transportation of frozen foods[3].

These studies demonstrated that commercially canned and frozen foods have retained substantial amounts of their nutrients. Products that are significant sources of nutrients when fresh, remain significant sources in their commercially processed forms. These two major research efforts provided most of the data on canned and frozen foods that have been incorporated in the current edition of the USDA Handbook No. 8, "Composition of Foods."

A few examples from the nutrition survey of canned foods will serve to recall some of these findings, which may already be familiar to several members of this audience.

This summary of ascorbic acid retention data is taken directly from "Canned Foods in Human Nutrition." It indicates the range of ascorbic acid retentions together with the mean value and the total number of observations for each product. In most instances, more than 70 percent of vitamin C is retained in the canned product. The variability of the retention is indicated by the length of the horizontal bar. There was relatively little variation in retention of ascorbic acid in asparagus and most of the canned fruits. In contrast, green beans, peas, and tomato juice exhibited a considerable range. This variation reflects some of the difficulties inherent in the conservation of this vitamin. It is susceptible to oxidative destruction during heating, and traces of copper from food processing equipment can act as a catalyst in this destructive process. As a result of the nutrition survey work, steps were taken in the industry to eliminate conditions in canning lines that might contribute to the destruction of this vitamin.

These data on carotene retention illustrate the minimal effect of thermal processing on this nutrient. More than 90 percent of the carotene content is conserved in most of the products. In the case of tomatoes and tomato juice, some carotene is discarded with waste peel and core material. The retention range in these instances is apparently due to variations in peeling and trimming operations before filling the can.

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[2] Sanborn, N. H. Conservation of vitamin C in tomato juice production. Canning Trade 60: 96. 1938.

[3] Cameron, E. J. The canning industry nutrition program. Food Technol. 8: 586. 1954.

While these data refer specifically to canned foods, they are intended to serve in an illustrative capacity for other forms of commercial processing. When prepared for the table, there are few dramatic differences in nutrient content between the various forms of the commercially processed and fresh products. These data taken from Bender (1966)[4] show the retention of ascorbic acid in peas after various stages of processing and after cooking. Fresh peas are compared with frozen, canned, air-dried, and freeze dried product. When ready for the table, the differences among these products are nutritionally insignificant.

In a presentation of this sort it is, of course, impossible to do more than provide a few illustrative examples from the enormous volume of data available. The nutrients provided by 100 gram servings of five canned vegetables are tabulated. The data come directly from USDA Handbook No. 8, but the bulk of the analytical values came from the National Canners Association-Can Manufacturers Institute nutrition program. In most instances, the analytical data on nutrient content were collected on crops produced during the 1942 and 1943 canning seasons.

With nutrient labeling just around the corner, and the reawakened interest in nutrition generated by the White House Conference on Food, Nutrition, and Health, a new round of questions about the nutritional values of commercially processed foods has begun. Some are generated by the simple fact that much of the data are about 30 years old. These are perfectly valid questions. Commercial varieties of some canning and freezing crops change with great rapidity. Since nutrient content in the raw product depends heavily on genetic factors, new varieties might have shifted in their nutritional values.

To repeat the analytical work on all the products included in the earlier survey would represent an enormous and perhaps unnecessary undertaking, especially at a time when the effort might be better spent on newer products on which data are scarce. To provide an indication of the nutritional content of the current commercial production of these crops, we have elected to sample certain products on a national scale utilizing sampling techniques strictly analogous to those employed in the 1942-43 surveys. As a first effort, tomato juice was selected as the product for a nationwide sampling program as follows:

PROXIMATE CONSTITUENTS – TOMATO JUICE

Proximate constituents and year	No. of samples	Average	Range	Standard deviation
		Percent		
Solids				
1969	130	6.47	5.3 to 7.9	0.78
1941 to 1943	164	6.3	4.8 to 9.0	-------
Protein				
1969	130	0.77	0.56 to 1.16	0.08
1941 to 1943	164	0.9	0.5 to 1.5	-------
Ash				
1969	130	1.22	0.95 to 1.56	0.11
1941 to 1943	164	1.1	0.7 to 1.6	-------

The analytical results obtained from the 1969 survey are compared with those from the 1942-43 seasons. The sampling techniques were as nearly comparable as we could make them, although in the case of some minor constituents we ran only a limited number of samples from the 1969 season.

The proximate constituents of the 1969 and 1941-43 tomato juice pack are quite similar. The total solids in the 1969 juice were somewhat higher but these could be the result of differences in climatic factors. The earlier survey was conducted over three seasons, while the present work was carried out on one season only. This is probably reflected in the narrower range of total solids in the 1969 samples, as compared with those from the three seasons' survey carried out earlier. There were trifling differences between the averages for protein and percent ash.

The remaining proximate constituents of the 1969 season averages somewhat higher in carbohydrates and was essentially identical in fat content and in total food energy, as shown below.

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[4] Bender, A. E. Nutritional effects of food processing. Jour. Food Technol. 1: 261. 1966.

PROXIMATE CONSTITUENTS – TOMATO JUICE

Proximate constituents and year	No. of samples	Average	Range	Standard deviation
		Percent		
Carbohydrates				
1969	130	4.36	3.1 to 5.5	0.55
1941 to 1943	164	4.0	2.4 to 6.8	------
Fat				
1969	37	0.05	0.03 to 0.10	0.02
1941 to 1943	164	0.1	0.03 to 0.30	------
Food energy (calories/100g.)				
1969	130	19	15 to 23	1.6
1941 to 1943	164	18	11 to 28	------

A comparison of minerals and trace elements is of some interest in connection with some recent speculation about a decrease in mineral and trace element content in the American diet as a result of increasing dependence on commercially processed food products. Detailed comparisons are not available for a large number of trace elements since only a limited number were included in the 1941 program. The national average for calcium in the 1969 season was 92 milligrams per 100 grams, as compared with 70 for the three seasons from 1941 to 1943. The ranges of the two periods were virtually identical. Comparisons are not available on magnesium, however, the phosphorous content of the 1969 juice was only slightly less than that of the earlier season. The difference would certainly be nutritionally insignificant, although the large number of samples available might provide a statistically significant difference.

MINERALS AND TRACE ELEMENTS – TOMATO JUICE

Constituent and year	No. of samples	Average	Range	Standard deviation
Calcium p.p.m.		Mg./100g.		
1969	129	92	35 to 110	31
1941 to 1943	138	70	30 to 110	----
Magnesium p.p.m.				
1969	129	101	67 to 142	74
1941 to 1943	------	------	--------------	----
Phsophorus p.p.m.				
1969	129	173	103 to 235	24
1941 to 1943	138	180	120 to 260	----
Iron p.p.m.				
1969	129	5.5	1.6 to 24.4	3.1
1941 to 1943	138	9	2 to 80	-----
Salt, pct.				
1969	59	0.82	0.43 to 1.20	0.13
Copper, pct.				
1969	75	1.04	0.31 to 4.90	0.63

The 1969 data indicate a slightly lower iron content. Iron in canned tomato juice products derives in part from the normal iron present in the raw tomato fruit, and partly as the result of container product interactions. Thus the difference in iron content shown by the two surveys could be either a result of differences in the raw product, or in the amount of iron dissolution from the walls of the container. The copper concentration in the last line of the following should be given as parts per million and not percent.

The more interesting comparisons are those shown below which summarize the results of the two surveys on ascorbic acid and carotene. These data also provide very little evidence for a marked change in the nutrient content of this product during the 26-year period since the last survey was completed. The national average vitamin C content of the 1969 juice was 13.4 mg. per 100 g. as compared with 14.4 for the three-season survey completed earlier. The range was not much different, although both the minimum and maximum values were lower than those of the earlier survey. The very low value of 1 mg. per 100 g. was obtained on a single sample and there is no obvious reason for the low result. The copper content of this sample is somewhat higher than average and this might have contributed to a loss of ascorbic acid in this one instance. The relatively wide range of vitamin C values in this product in both the 1969 and 1940 surveys is worth noticing. This degree of variation is not especially unusual. It poses potential difficulties in nutritional labeling, if requirements for accuracy are established that do not take into account normal, natural variations. Expressed in terms of a percent of a mean, the 5 milligram standard deviation represents about 37 percent of the average vitamin C content, as shown below:

VITAMINS – TOMATO JUICE

Constituent	No. of samples	Average	Range	Std. dev.
Absorbic acid				
		Mg./100g.		
1969	130	13.4	1.0 to 25.3	5.0
1941 to 1943	140	14.4	2.5 to 30.0	-----
Carotene				
		IU/100g.		
1969	130	690	376 to 1253	163
1941 to 1943	140	850	267 to 1600	------

Although not susceptible to heat destruction, the carotene content also exhibits a substantial range in both surveys. The 1969 survey average of 690 international units per 100 grams is somewhat lower than the previous value. In this instance the standard deviation of 163 international units represents approximately 24 percent of the average value.

To provide for a more uniform ascorbic acid content in tomato juice, the industry is seriously considering the possibility of fortification of this product. Vitamin C fortification has received a favorable reaction from most canners and it appears likely that a petition to amend the Standard of Identity for Canned Tomato Juice will be submitted in the near future.

At the National Canners Association laboratories our survey of the nutritional content of canned foods continues. Analytical work is in progress on a nationwide sampling of canned corn from the 1971 season. There has been very little analytical work on the nutrient content of canned corn since the major program of the 1940's.

In addition to this program in progress at the NCA Laboratory, many individual food producers are carrying out extensive analytical programs on their own products.

We expect that the Food and Drug Administration will soon publish additional information on the regulations implementing nutrient labeling. Although the exact form of these regulations is not yet clear, there will certainly be requirements relating to the essential accuracy of any statements concerning the nutrient content that are placed on the label.

For the processor of "natural" or unfabricated products similar to those we have been reviewing today, quantitative label statements concerning nutrient content may present a very difficult problem. Labels must be ordered a year or more in advance of production, and it is clearly not practical to predict all of the factors affecting nutrient content. A realistic nutrient labeling program must provide for normal product variation. That is, the requirements for labeling accuracy must not be such as to force the processors of unfabricated or "natural" products to make extremely low-label declarations in order to insure that their products are always in compliance with the regulations.

This would place them at a distinct disadvantage to producers of fabricated products, in which the level of the nutrient can be controlled by the manufacturer.

The White House Conference and the advent of nutrient labeling have resulted in a substantial increase in the expenditure of efforts and money on the collection of up-to-date nutritional data. Although most major food processors had maintained an active nutrient monitoring program as a part of their regular research efforts, there is no question that these efforts have sharply increased in the last 2 years. In the canning industry, we are reviewing several of the products produced in large volume to compare the nutrient content of current production with data obtained in the previous research program about 25 to 30 years ago.

We also need more detailed data on the nutrient content of formulated products, such as stews and specialty items. These have always represented certain difficulties in any industrywide nutrient survey program. Since formulations or product receipes can vary from one company to another, it is difficult to design an industrywide program which can take these differences into account. For such products, it will usually be necessary for individual producers to provide the nutritional data for their own products. This might be accomplished either by actual product analyses, or by calculation from reliable data on the product ingredients and a knowledge of the effects of formulation and processing on the nutrients.

Where adequate data are available for comparisons between the level of nutrients in today's production and those of 25 or 30 years ago, there is as yet no indication of any substantial decline in nutritional value when products are sampled on a national scale. The industry should, and certainly will, continue to monitor the nutrient content of its products. It will continue to make improvements wherever possible without unduly raising the cost to the consumer. The initial information available from these monitoring efforts, however, supports the conclusion that our processed food supply remains the same good nutritional bargain that it has always been.

SAFE PRACTICES OF HANDLING FOODS IN THE HOME

by
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Most people who buy and serve food for themselves and their families assume that all products in retail markets are safe, nutritious, and wholesome. Often they seem unaware that absolute protection is not attainable and they tend to underestimate their individual responsibilities for selective buying, proper home storage, hygienic food preparation, and sanitary kitchen practices. Such attitudes are understandable because modern technology has produced an abundance of high-quality, convenience foods that seldom, if ever, make people sick. For example, milk and ice cream, which were once major vehicles of disease transmission, can now be consumed without appreciable risk wherever Grade A pasteurized products are sold. Similarly, frozen and canned foods have excellent safety records, but they occasionally cause trouble when an unwary purchaser uses an abnormal product or abuses it in the home.

At present, insanitary food-handling practices are among the weakest links in the chain of food protection in the United States. The following discussion is intended to call attention to some measures that ordinary individuals can take to prevent foodborne illnesses in their homes.

RESPONSIBILITIES OF GOVERNMENT AND INDUSTRY TO THE CONSUMER

Both government and industry bear heavy responsibilities, not only to maintain the wholesomeness of the food supply, but to impress upon the individual consumer his role in the proper selection and preparation of everything he eats or drinks. To this end, a number of Federal agencies have published information intended for public education, and many of the available documents, including "Keeping Food Safe to Eat,"[1] are listed in Consumer Product

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[1] Human Nutrition Research Division, Agricultural Research Service, 1969. Keeping Food Safe to Eat — A Guide for Homemakers, U.S. Dept. of Agriculture Home and Garden Bulletin No. 162. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402 (10 cents).

Information[2], which is issued by the General Services Administration. However, the 1971 issue does not include several pertinent items from the U.S. Department of Health, Education, and Welfare such as "Hot Tips on Food Protection,"[3] "Cold Facts About Home Food Protection,"[4] "No Picnic,"[5] "From Hand to Mouth,"[6] and "You Can Prevent Foodborne Illness."[7]

Publication does not necessarily result in acceptance and use of the information by the individuals who need it most. The difficult problems of motivating consumers to prevent abuse of food were discussed at length during the 1971 National Conference on Food Protection in Denver. The Conference Proceedings include a report by Steuart[8] and others that describes the nature of the task, proposes a plan of action and recommends that local, State, and Federal agencies should participate in a continuing effort to focus public attention on self-protection against hazardous or unwholesome foods. Although the report is not entirely explicit about the techniques to be used, it offers a useful approach to the problem.

Any effort to improve food protection in the home must overcome a number of obstacles. First, family customs and personal habits tend to dominate day-to-day practices, regardless of the reasons for their original adoption or their current utility. For example, putting hot food in an old-fashioned icebox was recognized as an invitation to disaster because it melted the ice, and the food spoiled. This fact has been so firmly established that few housewives are willing to put hot food in an electric refrigerator, even though they know it will readily overcome the heat load and cool the food quickly.

Second, the advertising and labeling of commercial food products stress their good qualities and seldom, if ever, mention the microbial hazards or the nutritional limitations of the products. Furthermore, the publicity given to the work of food protection agencies at all levels of government emphasizes either the value of the protection offered or the successful legal actions taken to eliminate specific hazards. Relatively few consumers really understand the difference between quality grading and safety inspections, or the statistical and technological limitations of most surveillance programs. If a food product is under any kind of government inspection, the public seems to assume that there is no doubt about its safety. Although general experience tends to support this view for many products, it is an over-simplification that does not recognize the persistence of some contaminants or the many opportunities for recontamination after processing, distribution, or preparation of foods in the home.

A third obstacle to improving food handling practices in the home is the difficulty of demonstrating conclusively that insanitary practices are, in fact, responsible for the foodborne illnesses that do occur from time to time. There is an understandable reluctance to attribute illness to food because of the embarrassment suffered by those who prepared it. The problem is compounded by the fact that insanitary practices do not cause illness unless they occur in combination as shown below:

CONDITIONS THAT LEAD TO FOODBORNE ILLNESS

1. Presence of disease-producing agent.
2. Favorable food for agent survival.
3. Contamination of food with agent.
4. Persistence of agent during food preparation.
5. Serving contaminated food to susceptible individual.
6. Eating enough contaminated food to cause symptoms.

In order for food to become a vehicle for disease transmission, it must either already contain a disease-producing agent or be handled in a manner that will allow contamination from the cook or the surroundings. If contamination occurs, the food must provide an environment in which the agent can survive and perhaps proliferate in the final product as prepared for serving. Even then no illness will occur unless a susceptible individual eats enough contaminated food to take in a dose of the agent that will actually cause clinical illness. Because no one insanitary practice necessarily leads to illness, there is a tendency to disregard the potential importance of careless food handling unless it causes outright spoilage. The housewife can easily become complacent about her role in the prevention of foodborne disease because her mistakes do not necessarily satisfy all the conditions mentioned above for the occurrence of illness.

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[2] Consumer Product Information Coordinating Center, 1971. Consumer Product Information — An Index of Selected Federal Publications on How to Buy, Use and Take Care of Consumer Products, 2nd ed., GPO: 171-0-442-611. GSA Consumer Product Information, Washington, D.C. 20407.

[3] Public Health Service, 1966. Hot Tips on Food Protection. PHS Pub. No. 1404. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402 (10 cents).

[4] Public Health Service, 1968. Cold Facts About Home Food Protection, PHS Pub. No. 1247. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402 (5 cents).

[5] Public Health Service (no date). No Picnic. PHS Pub. No. 1623. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402 (5 cents).

[6] Public Health Service, 1966. From Hand to Mouth. PHS Pub. 281, 48 pp. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402 (20 cents).

[7] Public Health Service, 1963. You Can Prevent Foodborne Illness. PHS Pub. No. 1105. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402 (5 cents).

[8] Steuart, G. W., Hill, M., and White, P. 1972. Workshop 4: Education of the Consumer That Will Minimize the Abuse of Foods. Proceedings of the 1971 National Conference on Food Protection, pp. 92-114. Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402.

OCCURRENCE OF FOODBORNE DISEASE IN THE HOME

The prevalence of foodborne illness is difficult to determine because very few, probably less than 10 percent, of such outbreaks are officially reported to the public health authorities. Except for very severe cases, nonfatal gastroenteritis is seldom brought to the attention of a physician, and epidemiological studies are not initiated to demonstrate that a particular food was actually responsible for the illness. The best statistics currently available are from the Annual Report for 1970 of Foodborne Outbreaks, issued by the Center for Disease Control.[9]

The following tabulation summarizes the total outbreaks reported during 1969 and 1970 according to the places where the food was eaten, such as restaurants, homes, or schools. Of the 737 outbreaks, 289 (or nearly 40 percent) were associated with foods eaten at home; 243 (or about 32 percent) were associated with restaurants; and 64 (or less than 10 percent) were associated with schools. On the other hand, foods served at home accounted for only 3,321 cases of illness; whereas, the restaurants accounted for 14,496, and the school-acquired outbreaks accounted for 24,755 cases. Presumably, the distribution of cases relates to the number of people exposed, which is rather small in most families and tends to be large in restaurants or institutional feeding operations.

FOODBORNE ILLNESS ACQUIRED BY EATING AT VARIOUS TYPES OF SERVING OPERATIONS[1]
(1969-70)

Place of acquisition	Number of outbreaks	Number of cases
Restaurants, cafeterias, and delicatessens	243	14,496
Homes	289	3,321
Schools	64	24,755
Camps, churches, and picnics	38	3,350
Other	103	6,089
TOTAL	737	52,011

[1] Adapted from table 10, page 16, Foodborne Outbreaks-Annual Summary, 1970, Center for Disease Control, U.S. Dept. of Health, Education, and Welfare.

The report also analyzes the frequency with which different foods are associated with outbreaks. The three most commonly implicated types of foods in order of descending frequency are red meats, poultry, and seafoods. It is also interesting to note that fluid milk, which was once a major vehicle of disease transmission, now accounts for only two to five outbreaks per year caused by consumption of raw or improperly pasteurized milk.

The three leading types of illness were staphylococcal food poisoning, *Clostridium perfringens* poisoning, and salmonellosis, which together were responsible for more than half of all the outbreaks. The commonsense application of ordinary sanitary practices will prevent most of these foodborne diseases in the home, and the continued occurrence of outbreaks is largely the result of apathy, ignorance, poor judgment, or carelessness.

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[9] Center for Disease Control. Foodborne Outbreaks — Annual Summary, 1970, 35 pp. PHS, U.S. Department of Health, Education, and Welfare, Atlanta, Ga. 30333 (typewritten photocopy).

SELECTION OF FOODS IN RETAIL MARKETS

Examples of self-protective measures for grocery shoppers are shown below. Careful examination of each item may detect incipient spoilage, a torn package, an imperfect seal, or a bulged can. A quick look at the display cases will reveal whether (a) frozen foods are stacked above the frost line, (b) perishable products, such as milk or raw meat, are being held without refrigeration, and (c) delicatessen items, such as barbecued chicken or custard desserts are stored at temperatures that permit multiplication of bacteria. Any one of the above faults is sufficient reason to reject the product as potentially hazardous.

SELECTIVE GROCERY SHOPPING

1. Look for abnormalities and damage.
2. Observe display conditions.
3. Read the label.
4. Buy perishable food in small amounts.
5. Object to spoiled or inferior products.

Additional useful information may be obtained by reading the label carefully. A busy grocery store is not conducive to thoughtful reading, but examination of labels at home should reveal the ingredients, special precautions for storage, if required, and perhaps directions for serving. If the information seems deceptive, confusing, or inadequate, return the item or at least refrain from buying it again. Objections to a container, a display arrangement, a product, or its label should be called to the attention of the store manager. If a law appears to have been violated, the local health department or other appropriate control agency should also be notified. Unless the customer is willing to provide such information, neither the store management nor the government has a basis for corrective action.

A further precaution in buying food is to avoid stockpiling quantities that cannot be used within the normal shelflife of the product. The anticipated savings and conveniences from quantity purchases may be more imaginary than real. Losses due to spoilage, and possibly to the inconvenience of food poisoning, may be expected when perishable products are kept too long.

SAFE TRANSPORTATION AND HOME STORAGE

The care of food from the time of purchase until it is used can be an important factor in its preservation and safety. Some suggested precautions are given below.

TRANSPORTATION AND STORAGE OF FOOD

1. Minimize temperature changes in transit.
2. Keep in protective environment.
3. Exclude filth and pests.
4. Rotate stocks.
5. Check for deterioration before use.
6. Separate from household chemicals.

Incubation of a sackful of groceries from several hours in an automobile on a hot day hastens spoilage. If disease-producing bacteria should be present, they may also multiply and cause illness when the food is eaten. A safe shopping rule is to make the grocery store the last stop before returning home. On arrival each product should be stored promptly and continuously under the conditions best suited to its protection. Obviously frozen foods should be kept in a freezer at 0° F. or below, and perishable foods will require refrigeration at 45° or below, unless they are to be eaten immediately. Shelf-stable products, such as canned goods or dried foods, usually may be stored on a clean shelf in a cool dry cabinet. Perishable items are sometimes packaged in plastic bags, jars, or cans that resemble those used for shelf-stable products. If such a perishable product is inadvertently stored without refrigeration, it may become unsafe to eat because of bacterial growth, and it should be discarded even though no obvious spoilage is visible.

All food storage areas should be constructed to exclude filth and pests, especially insects and small rodents. A cabinet with a sewer pipe running through it is not a safe place to store food, because of potential leakage from the pipe and the difficulty of sealing the openings through which it passes. Frequent cleaning of storage areas and their contents is needed to remove dust that may contaminate the food with microorganisms when stored packages are opened for use.

A system of identifying the time of storage is desirable so that the oldest items will be used first. Most foods deteriorate gradually, even under good storage conditions and when kept beyond their normal shelflife, they frequently become inferior in nutritional and organoleptic qualities. If the container also deteriorates, the food may be contaminated from external sources.

All foods should be examined critically when being prepared for serving to detect abnormal appearance, odor, or physical properties that might indicate spoilage. Stored food should not be tasted before thorough cooking because some bacterial contaminants, such as Clostridium botulinum, produce potent toxins in concentrations that could cause death if a fraction of a spoonful were eaten. Any evidence of spoilage should be a signal to discard the food in some way that does not permit it to be eaten by animals, which might also be harmed by it.

Food storage areas should be used for no other purpose. A common cause of home accidents is the mistaken substitution of a household chemical for food ingredient because both happen to be stored in the same area and have a somewhat similar appearance. Cleansers, solvents, paints, polishes, and pesticides, as well as certain medicines and cosmetics, can be very poisonous when swallowed and should never be stored with foods.

SAFE CULINARY PRACTICES

The most important requirement for safe handling of food at home is continuous application of the basic principles of personal hygiene and food sanitation in the kitchen. The examples given below of sanitary culinary practices are so elementary that discussion of them with adults who can read and write may seem unnecessary. Nevertheless, failure to use these practices is responsible for more cases of foodborne illness than all other mistakes that are made in handling foods at home.

SANITARY CULINARY PRACTICES

1. Wash hands.
2. Use clean utensils and equipment.
3. Cook thoroughly.
4. Serve promptly.
5. Refrigerate quickly.
6. Thaw frozen food under controlled conditions.
7. Avoid repeated thawing and freezing.
8. Discard questionable foods.

The multiple demands of family life seem to create situations that invite disregard of safe culinary practices, unless the homemaker is highly motivated to maintain them. Consider the housewife who has just begun to prepare dinner when she realizes the baby's diaper needs changing, and at about the same time, she receives a telephone call from her husband saying that he is bringing a visitor home for dinner who must then go to the airport. It would not be surprising if she forgot to wash her hands or sliced a hard-boiled egg with the same knife she had used to open the package of frozen raw chicken. She might be tempted to stuff the chicken before it was completely thawed and then be forced by the visitor's travel schedule to serve the meal before the chicken was thoroughly cooked.

Her deviations from safe culinary practices could result in the illness of her family and guest, if the other conditions noted on page 100 were met. If not the contamination introduced during the preparation of her dinner could remain in the leftover food. Unless the food were promptly and continuously refrigerated, the microorganisms could multiply and be even more hazardous when eaten at another meal. The principal difficulty with making people realize the importance of such insanitary culinary practices is the unpredictable frequency with which other circumstances intervene to prevent illness from actually occurring. When an episode does occur, neither the homemaker nor her victims are likely to overlook or forget the event, but they may fail to associate it with the events that were responsible.

TIME-TEMPERATURE RELATIONSHIPS

Temperature control is the most effective measure available in the home to protect food against hazardous microbial contamination. The values given below are critical temperatures for the preservation and preparation of food for serving.

CRITICAL TEMPERATURES FOR SAFE FOOD HANDLING

Operation	Internal Temperatures °F.
Canning	240 to 260
Cooking	>165
Warm holding	>140
Dish washing	120 to 140
Danger zone	45 to 115
Refrigeration	35 to 45
Frozen storage	<0

The length of time that food is held at these temperatures is very important because the time-temperature relationship determines what happens to the microbial contaminants. Continuous storage of frozen food at 0° F., or below, prevents microorganisms from growing and kills some cells, but it does not destroy all of them. In addition, frozen storage retards chemical deterioration and effectively preserves the food for many months. When thawing occurs, microbial growth and chemical changes accelerate as the temperature rises. Unless thawed foods are used promptly, they will begin to spoil and may be hazardous to eat.

Refrigeration at 35° to 45° F. is very effective for short-term storage of perishable products. It slows down the growth of spoilage organisms and stops the multiplication of most infectious agents. The danger zone lies between 45° and 115°, where both infectious bacteria and toxin-producing microorganisms grow rapidly. In this zone, foods that offer favorable conditions of moisture, pH and available nutrients, may undergo a doubling of the bacterial population every 15 to 30 minutes. Exposure of perishable foods to temperatures in the danger zone should be avoided, if possible, or at least kept to the minimum time necessary for expeditious preparation and serving.

Cooking foods, such as pork roast or stuffed turkey, until the innermost part of the food mass reaches 165° F. will kill all infectious microorganisms, though it may not destroy the most heat-resistant spores or toxins. A thermometer should be used to determine the time required to reach this critical temperature because it will vary with the method of cooking as well as the kind and amount of food. Cooked foods should be served as soon as possible. When warm holding is necessary, the temperature should be kept above 140° to destroy post-cooking contamination and provide a margin of safety against holding in the danger zone. If cooked foods are to be kept for later use, they should be refrigerated immediately, without preliminary cooling. Distribution of the food in layers less than 4 inches deep will hasten the cooling process through the danger zone.

Fortunately, well-controlled commercial, canning processes have largely replaced home canning. The latter is an uncertain method of food preservation because reliable controls are seldom available to assure that the internal temperature of every can reaches 240° to 260° F. for any specified time. Underprocessed canned vegetables and meats are prone to develop lethal concentrations of botulinal toxin. As noted above, any evidence of swelling, decomposition, or other abnormality is sufficient reason to destroy the entire lot.

SUMMARY

Outbreaks of foodborne disease are more frequently associated with home-prepared foods than with commercially-prepared products or with items served in commercial eating establishments. The most commonly implicated foods are meat and poultry products and the leading types of illness are staphylococcal food poisoning, salmonellosis, and Clostridium perfringens poisoning.

Foodborne diseases are largely preventable in the home by the common-sense application of ordinary sanitary practices such as discarding abnormal or spoiled foods, using clean utensils and work surfaces, keeping warm foods hot and cold foods refrigerated, thorough cooking, and minimizing delays between preparation and serving. Mishandling of foods that allows the introduction, survival, and growth of disease-producing microorganisms is usually the result of apathy, ignorance, poor judgment, or carelessness. Any major improvement in the situation will require greater motivation of the many individuals who buy and serve food.

GUIDELINES FOR GOOD LABELING – DECEPTIVE ADVERTISING

by

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The Pillsbury Co.
Minneapolis, Minn.

My talk today covers two distinct areas that involve different skills and techniques, but are interrelated. The important thing to remember is that advertising and labeling should complement each other and together they should be informative to the consumer in a meaningful way. In recent years or even months advertising and labeling has become what educator Neil Jacoby would call a social problem created by a gap between society's present expectations of social conditions and the present realities.

Television and rising affluence have increased expectations. However the consumer has become far more critical and in many cases skeptical as to whether or not a product lives up to its claims. There is an increasing demand for proof, furthermore this campaign for truth has been pushed forward by the Federal Trade Commission (FTC) and rightly so. In the process of expanding their actions in the field of advertising they have developed new techniques of punishment such as corrective advertising — so that if you've done a wrong, a certain percent of future ads must tell the consumers that the previous advertising was false or misleading. Another possibility suggested is counter advertising — much as the anti-smoking ads we've all seen on TV. Probably the most far reaching and one which will probably have the greatest impact will be the requirement for substantiating or verifying and documenting data on claims in advance of any advertisements. A recent Supreme Court ruling will probably have far-reaching effects since it ruled that the FTC can go beyond the letter and even the spirit of antitrust laws in combating immoral or unfair business schemes. Since this type of advertising is detrimental to all business, I don't believe any reputable manufacturer would disagree with this ruling.

The factors I've just mentioned will require that a much tighter control be established within organizations on labeling and advertising. We have found that to accomplish this it is necessary that the climate within the organization be such that the individual can participate and be heard. I consider myself fortunate since the climate for this to happen has existed for many years within The Pillsbury Company. To give you some idea as to this climate, I'd like to quote excerpts from recent speeches by executives in our company.

Robert J. Keith, Chairman of the Board of the Pillsbury Company, recently gave a talk about consumerism which certainly is a vital part of developing adequate response and communications. He stated "consumerism is a social force within the environment designed to protect the consumer by exerting legal, moral, and economic pressure on business. Consumerism is here. It is not a passing fad. Its positives and negatives influence more and more purchases of goods and services . . . not to mention influencing legislation, academic curricula, religious sermons, union-management relations and negotiations, technical and scientific pursuits, and social conversations." He further stated "if consumerism is to become a dominant value in our system . . . and it must or our system will disintegrate . . . the motivation must come from the management. It is the duty of management to police ambivalence, anachronism and dichotomy in the company's policies and actions . . . to put it a bit more bluntly, the conscience of a corporation must be spelled out by the leaders." I might add that Mr. Keith is also Chairman of the Sub-Council on advertising and promotion of the President's National Business Council for consumer affairs.

Terrance Hanold, President of Pillsbury, in an address to a number of the Nation's food editors concerning the principal issues with which the food industry must deal, stated "Increasingly it is clear that the industry must either provide leadership in dealing with these problems or become itself the hard core of the problems. We can lead or impede, but we cannot stand aside."

But even though the climate is right in the organization, we've found that it's just not all that easy to communicate with the consumer — especially technical information — for instance in nutritional labeling. We find that we are handicapped by a vocabulary that has been designed by and for the use of nutritionists and other professionals. We're going to have to somehow build in information such as the purpose of additives and the role certain nutrients play in the diet into the label and other forms of communication with the consumer.

There are a number of publications available published by the National Academy of Sciences, U.S. Department of Agriculture, The Dairy Council and others on nutrition that could help all of us in this area. Unfortunately these are not known to the general public. Information of this sort must be part of a nutritional education program and should ultimately allow for the designation of say Vitamin C instead of ascorbic acid, Vitamin E instead of a tocopherol acetate, and Vitamin D rather than irradiated ergosterol---. Unfortunately in attempting to do this we begin to tread in gray areas in that someone might decide that simple terms might conflict with the intent of the label laws and be considered deceptive. Unfortunately, we have not developed a language such as the medical profession uses in communicating with the public. They don't tell a patient that he has a myocardial infarct; they tell him he has had a heart attack.

The problem of language and informative communication is very difficult and because of this a number of nutritional labels are being evaluated with consumers, sponsored by industry and Food and Drug Administration (FDA). From this effort hopefully we will have a format for nutrient labeling that will be compatible with an education program and will be understood by all consumers. To emphasize how difficult this is, we have found in research studies that most consumers are aware of nutrition but are lacking in knowledge for instance as to the types of foods that carry certain nutrients. We have found, to name a few, that the things the housewife knows best are as follows:

Calcium comes from milk and builds bones and teeth, but when they are asked the principal benefits of milk, vitamins rank first. There is a low level of knowledge of milk as a source of protein which is shocking when we consider the level of advertising by the milk industry.

Meats and poultry provide protein which builds muscles and provides strength.

Orange juice provides vitamins which prevent colds.

Sugar provides quick energy. It's fattening and bad for the teeth.

Iron builds blood — comes from liver — other sources were not known.

Potatoes and baked goods are in the same image. The perceived nutritional benefit is primarily to be filling. There is very little knowledge of true nutritional benefits and a high concern with these items being fattening.

Fats in the form of most dairy products, except milk, have no clear nutritional benefit, are bad for you because they are fattening and contribute to heart disease.

Breakfast cereals provide vitamins which make one healthy.

Vitamins are good for you in a vague way — the only specific benefits are orange juice for cold preventions and carrots for good vision.

The benefit of these important and economical foods is downgraded by half-knowledge. These opinions are apparently shared at all social and income levels. I believe these few examples further illustrate the magnitude of the job of educating people in nutrition.

In the field of advertising, I think an additional quote from Mr. Keith's speech sums up the problem: "It is my belief that the manufacturers and retailers who spend millions of dollars on advertising are getting some just criticism from the consumerist. It is my firm belief that we who manage the manufacturing and retailing enterprises of this country can eliminate this kind of aberration provided we move our advertising message from the psychological to the informative."

The informative part, we find, is quite difficult in visual and audio advertising. This problem is not as simple as just talking about a product in a TV or radio commercial. Albert Mehrabian, a U.C.L.A. psychologist, conducted controlled research which indicated that of the total message transmitted by TV newsman, only 7 percent was transmitted by words; of the remainder, 38 percent was by vocal intonation and inflection, and 55 percent by facial expression and physical posture. This kind of data indicates that we must be especially careful of how we use audio advertising or it might appear deceptive. We feel that audio advertising such as TV and radio should probably highlight certain key elements and then inform consumers where to find additional information — which is probably best carried on the label.

Labeling and advertising will be even more difficult in the future since as I look ahead into the future of the food supply in the United States, I am more and more convinced that many foods as we now know them will be changed. Many natural products will no longer be available because of economics. Other natural foods will have to be taken apart and toxicants removed from them. This indicates to me that we are entering an era of food types and manufacturing that makes the source of nutrients (whether it is cereal, meat, fruit, or vegetables) unimportant. The important factor will be the quantity and quality of the nutrients used in the fabrication of the foodstuffs. However, as we depart from the traditional composition, we will find the task of communicating what the product is and does and what it contains much more difficult, if not downright confusing — and unless done with extreme caution and sufficient testing for interpretation, might be considered misleading or deceptive.

While I'm on the subject of advertising I'd like to state my feelings that every company should have a written advertising policy — endorsed by the executive office of the company.

We do have such a policy in the company for which I work and I'm proud of it because I think it's a good, well thought-out policy that if followed can only result in factual information to the consumer. I'm also proud of the fact that insofar as is humanly possible it is being adhered to in all areas of the company — I'd like to go over it.

PILLSBURY'S ADVERTISING POLICY

The Pillsbury advertising policy has its philosophy rooted in the recognition that a commitment to the consumer's rights ("consumerism") is essential to the corporation's well-being.

Among these rights are:

The right to protection against fraud and deceit;

A guarantee of safe and healthful products;

The right to know the attributes of the products;

The right to freedom of selection among product alternatives with clearly defined price-value relationship.

It is our policy that every claim we make regarding the product advertised be substantiated and documented before use.

Pillsbury communications must reflect a sensitivity to consumer knowledge, sophistication, and skepticism. More specifically, a working policy should include the following:

1. We must insure that advertising and publicity materials not include "evidence" of any product attribute and benefit, when such evidence is not a true indication of the specified product characteristics. Thus, food products cannot be shown or written about in such a manner so as to exaggerate, misrepresent or in any way incorrectly show the product qualities. This applies to depiction of the product itself, as well as the reported results of any test, experiment or demonstration.
2. We must examine any piece of communication that could have a real or implied, purposeful or accidental, element of fraud or deception. Moreover, it is not good enough to simply look at a piece of advertising for the words or claims used. We must also read between the lines in search of elements which may lead to consumer misinterpretation.
3. We must insure that our advertising be in good taste. It cannot be offensive to any religious, ethnic, or political segment. Advertising must be competitive in a positive sense, not derogatory to the products of other companies.

Our policy thus demands a belief and conviction that we can have honesty and good taste in our advertising, and, at the same time achieve a level of creativity and credibility unattainable with advertising that reflects lower standards. We truly believe that high ethical standards for advertising are totally consistent with goals in the area of share of market and profit before taxes.

Further, this policy is backed up by procedures and pinpointing of responsibility relating to this policy, all the way from label approval to approval of all advertising and photography used in advertising. This system of course inevitably slows things down but it also helps us get away from the old axiom that "there's never time to do it right, but always time to do it over."

It means the scientists not only become involved in advertising material, but have a high level of responsibility for approving its accuracy. The scientists also have the responsibility for making sure that any necessary data is obtained which must also include the safety of the product under its intended conditions of use.

To give you some idea of what is involved before final label or advertising approval, I'll list some of the check points we have instigated.

Research and Development

Packaging — functionality, lack of slack fill or other deceptive packaging practice.

Formulation — accuracy of ingredient itemization, accuracy of declared net weight.

Ingredient control — compliance of formulation with applicable food additive regulations, proper names and order of ingredients.

Product safety office — safety of product for human consumption under ordinary conditions of use, freedom from advocacy or suggestions for misuse of product, and presence of any necessary warnings.

Vice president-science and technology — medical accuracy of all nutritional claims, safety and efficacy of nutritional products under recommended conditions of use.

Nutrition committee — The Nutrition Committee shall review the label for accuracy of nutritional claims and for communication thereof to the consumer without deception.

Quality assurance — ability of production facility to produce the product on a day-to-day basis in conformity with all labeling claims; presence of any required opening instructions, handling or storage instructions, open dating information, and coding.

Consumer service — accuracy of recipe copy, accuracy of yield statement, and integrity of pictorial representations.

Law department —

(i) General — truth of promotional copy and integrity of pictorial representations, and correspondence of label to all federal and state legal requirements. The Law Department may require, as a condition to giving its approval, that the Vice President-Science and Technology has secured written approval from the company's nutritional advisers of designated nutritional claims.

(ii) Patent and trademark counsel — correct use of registered marks, availability of marks used, lack of misuse of trademarks, accuracy of patent number on packages and noninfringement of existing patents or trademarks owned by others.

Procurement — that all necessary approvals have been obtained and that there are no apparent defects in the keyline.

And finally the Brand Manager delivers complete approved keyline to Procurement. Such delivery constitutes a representation by the Brand Manager (i) no changes in the keyline have occurred after an approval has been affixed to the keyline, or (ii) all those who approved the keyline prior to changes made in it have been notified in writing of the changes and have assented to the changes, or (iii) where a required approval is missing from the keyline a person authorized to give the missing approval has been informed in writing of the form and content of the keyline and has assented to it.

Procurement shall submit to label suppliers only those keylines which contain all necessary approvals.

For advertising —

The approvals are as follows:

Brand manager — general assembly of keyline, truth of advertising copy, and integrity of any pictorial representation.

Product safety office — freedom from advocacy or suggestions for misuse of product, and presence of any necessary warnings.

Vice president-science and technology — (where applicable) medical accuracy of all nutritional claims made in advertising, verified by expert consultants.

Nutrition committee — (where applicable) accuracy of nutritional claims and communication thereof to the consumer without deception.

Quality assurance — production can meet all advertising claims on a day-to-day basis.

Consumer service — integrity of pictorial representations, accuracy of product claims, and practicality of recipe suggestions.

Law department —

General — truth of advertising copy and integrity of pictorial representations, and correspondence of advertising to all federal and state legal requirements. In giving such approval, the Law Department will see that all necessary approvals have been obtained and will give due consideration to all approvals. The Law Department may require, as a condition to giving its approval, that the Vice President-Science and Technology has secured written approval from the company's nutritional advisors of designated nutritional claims.

Patent and trademark counsel — correct use of registered marks, availability of marks used, lack of misuse of trademarks, noninfringement of existing patents or trademarks owned by others.

Procurement — that in the case of point of sale and trade materials all necessary approvals have been obtained.

Then the Brand Manager forwards to the agency only those keylines, scripts and storyboards which bear all necessary approvals.

The Agency produces commercials involving television photography under the supervision of Pillsbury Consumer Service.

The Agency forwards Brand Manager copies of all television commercials prior to broadcast.

Brand Manager secures final written approval of all television commercials from Consumer Service and the General Law Department prior to first broadcast. In addition to the approval matters approved by the General Law Department and Consumer Service, such approval shall extend to the integrity of the video representations in the commercial.

There are other facets, such as advertising to children, etc., but I believe what I've covered is illustrative of the care necessary from product concept to market place.

The procedure I've just covered was developed by Earl Clasen, our Vice President of Marketing. I think that the clarity and explicit nature of the document reflects his years involved in the advertising of products and his concern for our integrity — and the fact that this document has been accepted at all levels in the Corporation indicates an overall reflection of concern. I would also like to state that as far as we are concerned the procedure is not inhibitive, but allows for greater interaction between all peoples involved and results in a greater level of creativity in meeting the rising level of consumer expectations.

Before closing I would like to mention that there are currently unofficial guidelines published relating to nutrition and nutrition labeling. A joint policy statement was issued in September of 1969 by the Food and Nutrition Board of the National Research Council and recently a policy statement on nutrition labeling of The American Dietetic Association was published in their Journal, Volume 60, No. 3, March 1972. The official policy, of course, will be those issued by The Food and Drug Administration. However, we should all keep an open mind and be willing to change if necessary as more consumer information is gathered. After all, the consumer should have a great deal to say about our communications.

SOME ASPECTS OF THE MEAT AND POULTRY PROGRAM APPLICABLE TO THE MODERN CONSUMER

by
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Introduction

Ralph Nader and his principles are not new phenomenon in this "Age of the Consumer." In 1221 Italy enacted a meat inspection law which provided severe punishment for butchers who slaughtered sows or boars or who sold animals that had died a natural death or who held meat over 24 hours without labeling it as such. The first offense resulted in a fine — one gold lire. The second resulted in the cutting off of the hand, and the third offense was punishable by hanging.

If these conditions of punishment were still applicable today, Louisiana would have had few butchers alive by the middle of 1970. Modern-day consumers do not expect violators of meat inspection laws to be so severely punished. However, they feel, and rightly so, that they are entitled to clean, wholesome, properly labeled products. In Louisiana, the meat consumer was provided this protection by the enactment and funding of a meat inspection law in 1969, which was amended to include poultry inspection in 1970. This Louisiana law came about because the Congress of the United States passed the Wholesome Meat Act in 1967 which allowed States 2 years to develop a State-inspection program.

Scope of Inspection Program

At present, our department inspection program requires that all animals presented for slaughter undergo an ante-mortem inspection. If the animal is healthy, it is slaughtered; however, if any question exists in the inspector's mind, a veterinarian is used to decide the final disposition.

Following slaughter, the meat carcass is subjected to a thorough post-mortem inspection which includes inspection of vial lymph nodes, viscera, and carcass. These inspection procedures are carried out in a facility which is acceptable sanitation, equipment, and construction-wise.

Processing and Manufacturing of Edible Products

After the carcass has passed inspection, the meat is ready to be processed. Processing is the use of this inspected meat in the manufacturing of edible by-products for human consumption. It also involves any cutting or boning. This branch of inspection presents problems to the inexperienced inspector. One problem encountered is the difficulty in distinguishing aged meat from unwholesome meat. Facilities used to process meat products must also be maintained in acceptable sanitary conditions. The final step of inspection is the branding or labeling of finished product to insure the consumer that this product has met acceptable standards.

Poultry Operations

Since 1970, poultry plants are also required to conduct ante-mortem and post-mortem inspections.

Poultry slaughter facilities are required to meet equally rigid sanitation standards. After inspection, chickens can be sold whole or cut-up into a variety of meat products.

In addition to state inspectors, each of the Louisiana Poultry Plants are staffed by USDA graders.

Federal-State Correlation of Program

Our inspection program is financed by equal State and Federal monies. Federal personnel can be called on at anytime to lend assistance on technical problems. Additionally, they are required to do annual reviews of all State-inspected plants. Furthermore, they assist our program by providing their training facilities when necessary. The major part of our training is conducted at the Baton Rouge training center and on the job.

Additional consumer protection is provided by our laboratory in Baton Rouge, located on the Louisiana State University Campus, which routinely runs sample analysis of meat products to check for improper formulations and residues of chemicals or biologicals.

Conclusion

Even though the Louisiana Inspection Program has been in existence only 2-1/2 years, I feel that the consumer can now purchase Louisiana meat products with the confidence that these products are wholesome. The inspectors we employ are being trained with you, the consumer, in mind.

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Bourdette, Vernon R., Information Officer
Burton, Gloria C. (Mrs.), Director's Office

Calamari, Timothy A. (Dr.), Cotton Finishes Laboratory
Catalano, Edwin A., Food Crops Laboratory
Codifer, Louis P., Jr., Oilseed Crops Laboratory
Conkerton, Edith J. (Mrs.), Oilseed Crops Laboratory
Connor, Mary Louise D. (Mrs.), Director's Office
Corkern, Ray S. (Dr.), Economic Research Service

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